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TRANSACTIONS

OF THE

ASSOCIATION OF AMERICAN PHYSICIANS.

FIFTH SESSION,

HELD AT WASHINGTON, D. C.,

MAY 13, 14, AND 15, 1890.

VOLUME V.

PHILADELPHIA:
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1890.

NOTICE.

THIS volume of TRANSACTIONS contains the papers read before the Association at the meeting held May 13, 14, and 15, 1890.

The Association assumes no responsibility for the statements and opinions published in this volume.

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MINUTES

OF THE

FIFTH ANNUAL MEETING

OF THE

ASSOCIATION OF AMERICAN PHYSICIANS,

Held in the Army Medical Museum and Library Building, Washington, D. C., May 13, 14, *and* 15, 1890.

The following members were present:

From New York: Drs. A. Brayton Ball, C. L. Dana, Francis Delafield, William H. Draper, A. Jacobi, Francis P. Kinnicutt, Alfred L. Loomis, William T. Lusk, George L. Peabody, J. West Roosevelt, M. Allen Starr, W. Gilman Thompson.

From Philadelphia: Drs. Henry F. Formad, John Guitéras, I. Minis Hays, S. Weir Mitchell, John H. Musser, William Pepper, Edward O. Shakespeare, Wharton Sinkler, James Tyson, James C. Wilson.

From Washington: Drs. John S. Billings, Samuel C. Busey, Robert T. Edes, W. W. Johnston, D. W. Prentiss.

From Baltimore: Drs. I. E. Atkinson, Samuel C. Chew, William H. Welch.

From Boston: Drs. Reginald H. Fitz, Charles F. Folsom, Francis Minot.

From Montreal: Drs. George Ross, James Stewart, George Wilkins.

From Chicago: Drs. Norman Bridge, Hosmer A. Johnson, Henry M. Lyman.

From Albany: Dr. Henry Hun.

From Aiken, S. C.: Dr. W. H. Geddings.

From Brooklyn: Dr. B. F. Westbrook.
From Chattanooga: Dr. James E. Reeves.
From Cincinnati: Dr. F. Forchheimer.
From Jamaica Plain: Dr. Harold C. Ernst.
From Portland: Dr. Israel T. Dana.
From Saranac Lake: Dr. Edward L. Trudeau.
From St. Louis: Dr. G. Baumgarten.
From Toronto: Dr. J. E. Graham.

MAY 13TH.—MORNING SESSION.

PRESIDENT BUSEY called the Association to order at 10.35 A.M., and, after having stated that the Minutes of the last annual meeting would be accepted as printed in the fourth volume of the *Transactions*, delivered the following opening address:

GENTLEMEN: In view of the fact that the consideration of important business will immediately engage your attention I have concluded that I could best economize time, and fully discharge the formality of an opening address, by a brief reference to the revision of the plan of organization proposed by the Council and submitted at the last annual meeting. In doing so I will have occasion to refer to the history and work of the Council during the past four years.

An association having for its object the advancement of scientific and practical medicine needs but few rules and regulations, and only such as will secure uniformity of procedure and continuity of purpose. Medical politics and medical ethics find no place in such an organization. The single purpose of scientific pursuit secures decorum without the aid and enforcement of parliamentary law and restraint. We are here to teach and to learn, and the honors fall to those who contribute most to the common stock of knowledge.

The proposed revision seeks condensation, brevity, and simplicity by a rearrangement of the articles and provisions of the existing Constitution, and promotes efficiency by a reconstitution of the membership and enlargement of the powers of the Council, and by a more precise definition of the duties of the officers. In fact, it conforms the plan of organization to the practical workings of the Association since its first meeting in this city, in June, 1886.

The Constitution, as it is, provides for a Council of seven members,

presumably residents of different—and, as has happened, remote—localities, and charges it with only so much of the important duties pertaining to the scientific interests as relates to the selection of "subjects for debate," but leaves it without an organization, and distinctly separates it, as a continuous committee, from the officers, whose duties make their coöperation and presence at its meetings absolutely necessary to promote the best interest of the Association and to secure a satisfactory discharge of the stated and implied duties of the Council. This defect was discovered by President Mitchell, who assumed the responsibility of summoning the seven councillors and all the officers in a general conference in the city of Philadelphia, thus establishing a precedent which has been followed by his successors with such satisfactory results. The proposed amendments will legalize this method of procedure, and reconstitute the Council by making it consist of the President, Vice-President, Secretary, Treasurer, Recorder, and five Councillors, and charge it with the duties of making all the arrangements for the annual meetings and of general supervision of the business affairs of the Association. It thus brings together the officers proper and the Councillors into one compact and continuous committee, thereby securing continuity of work and avoiding the delays, irregularities, and incongruities necessarily incident to the complete annual change of the *personnel* of the working and business committee.

Those of us who have served continuously upon the Council, as constituted by Dr. Mitchell, know something of the labor performed and of the sacrifices made by some of its members in leaving their business and travelling long distances to attend its meetings at seasons of the year when time was most valuable to them. The selection of subjects for debate and of referees and co-referees is a duty of the highest importance, but if the Council rested its labors at that point the programmes of the annual meetings would be made up of voluntary contributions offered by the few who would venture, without solicitation and appointment, to prepare essays.

The purely voluntary system of providing papers for the annual meetings of medical societies in vogue in some of them has proved satisfactory for a few years, but sooner or later the programmes of such bodies are, for the most part, made up of hastily prepared essays, the titles of which are sometimes not even determined until the time has arrived for their announcement, and of clinical reports of cases

loosely thrown together, without such analysis and comment as would make them valuable contributions to medical literature. The uniform custom of the Council has been to assemble soon after the adjournment of the annual meetings and to proceed at once to prepare for the next ensuing meeting by the selection of subjects for debate, referees and co-referees, and the designation of members who should be requested to prepare papers on such subjects as might be suggested or they might select, and also to invite, by a general announcement, voluntary contributions. The *Transactions* of the past four years and the programme of the present meeting attest the wisdom of this method of procedure. Its success, however, requires careful consideration and a knowledge of what has transpired. These duties, together with the examination into the qualifications of the nominations, impose much labor, which has been cheerfully and, I believe, satisfactorily performed. The continuous service of a competent secretary is an important, if not an essential, factor of success, and whilst the revision reserves the right to change that officer annually, the rule of continuous service has been established by the successive re-election of the present most efficient and capable officer.

This revision will make the President practically the chief executive officer of the society, and, by implication, holds him in a measure responsible for the scientific success of the meeting. At present he is simply a presiding officer decorated with the formality and privilege of making an opening address.

There is at present no authorized supervision of the business and financial affairs of the Association. These may be matters of minor importance, but monetary embarrassments and business complications have wrecked more than one scientific body. Prudence, if not necessity, suggests the propriety of an amendment which will impose this duty upon the Council.

To the minor details of the proposed revision I need not refer; only such have been suggested as will condense and simplify the plan of organization.

It remains for me to announce officially the death of one of the original and most distinguished members of the Association.

James Howell Hutchinson, of Philadelphia, an original member of the Association, died December 27, 1889, aged fifty-five years. Dr. Hutchinson possessed the confidence and respect of the community in

which he lived to a degree that is enjoyed by few men, holding many offices of medical and public trust, among the latter that of Trustee of the University of Pennsylvania. He was Vice-President of the College of Physicians of Philadelphia, and Physician to the Pennsylvania Hospital and to the Children's Hospital. Dr. Hutchinson was not a voluminous writer, but what he published was carefully composed, pure in style, and bore the marks of sound judgment and experience. His article upon "Typhoid Fever," in the *System of Medicine by American Authors*, edited by Pepper, and that upon "The Management of the Convalescent Stage in Typhoid Fever," read before this Association at its meeting in 1888, may be named as types of his literary work. As a practitioner of medicine, consultant, and hospital physician he was admirable; accurate in diagnosis, careful in prognosis, and his treatment, governed by sound sense and extensive knowledge, was surpassed by few. He will long be gratefully and respectfully remembered by the younger men who served under him in the hospitals with which he was connected as physician. The loss of such a man to any community is very great, and few are regretted as Dr. Hutchinson is, for he was a trustworthy physician, an honorable gentleman, and a straightforward, truthful man.

The Secretary read the following communication from the Secretary of the Executive Committee of the Congress of American Physicians and Surgeons:

NEW YORK, March 13, 1890.

HENRY HUN, M.D.,

Secretary of Association of American Physicians.

DEAR DOCTOR: I beg to call your attention to the following extracts from the Minutes of the Executive Committee of the Second Congress of American Physicians and Surgeons, which are furnished by direction of the Committee for your information as Secretary of the above-named organization:

The officers of the Congress are as follows:

President—Dr. S. Weir Mitchell, of Philadelphia.

Secretary—Dr. W. H. Carmalt, of New Haven.

Treasurer—Dr. J. S. Billings, of Washington.

Chairman of the Executive Committee—Dr. William Pepper, of Philadelphia.

Chairman of the Committee of Arrangements—Dr. S. C. Busey, of Washington.

The Congress will be held in Washington, D. C., on Tuesday, September 22, Wednesday, September 23, Thursday, September 24, and Friday, September 25, 1891.

The mornings of these days will be devoted to the special work of the various participating associations and societies, and the sessions of the Congress will occur from 3 to 6 o'clock in the afternoon of each of the above days.

The Presidential Address will occur on the Wednesday evening of the Congress.

A registration fee of $5 to each person will be charged, to be collected by the officers of the Congress.

Four subjects were selected for presentation to and discussion before the Congress, the exact phraseology of the titles to be decided by the officers of the Congress and the Chairman of the Executive Committee:

1. The relation of microörganisms to inflammation and suppuration.
2. The late manifestations of syphilis.
3. A diathetic subject, including heredity.
4. The influence of soils in the causation of disease.

The following resolutions were adopted:

1. "That foreigners of distinction shall be formally invited to attend the Congress, through the President of the Congress and the Chairman of the Executive Committee; and the various societies participating in the Congress shall be requested to furnish these officers with a list of those whom each society wishes to invite."

2. "That participating societies shall be privileged to accredit visitors to the Congress, who shall pay the registration fee, but shall not be privileged to participate in the deliberations of the Congress."

3. "That when the *Transactions* of 1891 are published the names of all visitors appear in the volume."

At the meeting held February 12, 1890, Dr. S. C. Busey, of Washington, D. C., was elected to represent your Association upon the Committee of Arrangements.

Respectfully yours,

NEWTON M. SHAFFER, *Secretary.*

Dr. Jacobi, Chairman of the American Committee of the Tenth International Medical Congress, extended an earnest invitation to all the members of the Association to attend the Congress. He also explained that the American Committee was appointed by the German Executive Committee at an unfortunately late date in the formation of the Congress, and that this American Committee is composed almost entirely of physicians living in Eastern and Southern cities, because circular-letters of invitation to join the committee, sent to physicians in Chicago, St. Louis, and other Western cities, were either not answered at all or were answered at too late a date.

The following revised Constitution was then adopted by each article in turn and as a whole, it being understood that no Councillor should be elected either at this meeting or the next until the number of Councillors should be reduced from seven to five, as therein provided.

Article I.

Object and Name.

This Association has for its object the advancement of scientific and practical medicine, and shall be known as The Association of American Physicians.

Article II.

Members.

The number of active members shall be limited to one hundred. Physicians of sufficient eminence to merit the distinction may, to a number not exceeding twenty-five, be elected honorary members, and as such shall be entitled to attend all meetings and to take part in the proceedings, but not to vote.

Membership shall lapse by reason of absence from three successive annual meetings without reason given acceptable to the Council. An excused absence shall not be regarded as an attendance. Membership may be forfeited for reasons deemed sufficient by the Association.

Article III.

Officers.

The Officers of the Association shall be a President, a Vice-President, a Secretary, a Recorder, a Treasurer—all to be elected annually;

five Councillors—one to be elected each year for a term of five years; and a Representative to serve on the Executive Committee of the Congress of American Physicians and Surgeons, to be elected at the annual meeting immediately following the Congress.

Article IV.

Duty of Officers.

The duties of the President, Vice-President, Secretary, and Treasurer shall be those usually assigned to these officers. The Recorder shall secure the papers read and also proper notes of the discussions thereon, and shall publish the annual volume of Transactions. The five Councillors shall constitute a Committee of Nomination of Officers, and as such shall present a report at the commencement of the morning session of the last day of the annual meeting, at the conclusion of which session the election shall be held by ballot. The Representative on the Executive Committee of the Congress shall have full power to represent the Association on all ordinary questions, but must be authorized by the Council of this Association before assenting to the admission of any Society to the Congress.

Article V.

Council.

The officers of the Association shall constitute the Council, of which four members shall constitute a quorum. It shall be the duty of the Council to supervise the affairs of the Association, to make all the arrangements for the annual meeting, to suggest the subjects for debate, and to consider the nominations for membership and report on them at the meeting at which they shall be balloted on.

Article VI.

Meetings.

1. The Association shall hold an annual meeting in the city of Washington, D. C., at a time designated by the Council.

2. The President shall call the meeting to order and open the session by an address not exceeding half an hour in length. In his

absence the Vice-President shall preside, and in the absence of both these officers, the senior Councillor.

3. Then shall follow original communications, discussions on subjects of general interest in the department of medicine and pathology, demonstrations in pathology, and miscellaneous business.

4. Authors of papers, and referees and co-referees who open a discussion shall not occupy more than thirty minutes each; and in the discussion following, the remarks of each speaker shall be limited to ten minutes.

5. The referees, co-referees, and authors of papers shall send abstracts of their papers to the Council for distribution to members previous to the meeting.

Article VII.

Quorum.

Any number of members present at the appointed time of the annual meeting shall constitute a quorum for the transaction of ordinary business, but for the election of members, fifteen shall be necessary for a quorum; and for altering the Constitution, twenty-five members shall be necessary.

Article VIII.

Election of Members.

Nominations to membership, signed by two members who are not officers of the Association, must be made in writing at an annual meeting of the Association; and within six months of the time of nomination the nominators shall transmit to the Council letters stating the qualifications of the nominee and also a list of his professional positions and publications. The Council shall report those nominations which are approved by it to the next annual meeting for action. An affirmative vote of three-fourths of the members present shall be necessary for the election of active members, but for the election of honorary members an unanimous vote of those present shall be required.

Article IX.

Annual Dues.

The Annual Dues of the Association shall be fifteen dollars, from the payment of which honorary members shall be exempt.

Article X.

Amendments to Constitution.

Amendments to the Constitution must be proposed in writing by five members at an annual meeting and must be acted upon at the succeeding annual meeting, the notices for which shall contain an announcement of the proposed amendments; and such amendments shall require for their adoption an affirmative vote of three-fourths of those present.

Dr. Reeves then read a paper on the "Natural History of Typhoid Fever," which was discussed by Drs. Wilson, Loomis, Mitchell, Johnson, Jacobi, Pepper, and Reeves.

Dr. Bridge then read a paper entitled "Inflammations of the Appendix and Cæcum, and the Duty of the Physician regarding them," which was discussed by Drs. Atkinson, Fitz, Jacobi, Pepper, Johnson, Musser, and Bridge.

Adjourned at 1.15 P. M.

Afternoon Session.

Association called to order at 3 P. M. by Ex-President Minot.

Dr. Pepper read a paper on "Varicose Aneurism of the Arch of the Aorta."

In the absence of Dr. Danforth, his paper on "The Diathetic Causes of Renal Inadequacy" was read by title.

Dr. Lusk read a paper on "Antisepsis in Midwifery," which was discussed by Drs. Welch, Jacobi, Pepper, Roosevelt, and Lusk.

Dr. I. T. Dana then read a paper entitled "Seizures Characterized by Shock and Loss of Consciousness," which was discussed by Drs. Lyman, Loomis, Edes, Jacobi, Tyson, and Dana.

Adjourned at 5 P. M.

May 14th.—Morning Session.

President Busey called the Association to order at 10.20 A. M.

Dr. Mitchell, as Referee, opened the discussion on the "Disorders

of Sleep," and was followed by Dr. Folsom, the co-referee, the discussion being continued by Drs. Pepper, Lyman, Peabody, Sinkler, Westbrook, Starr, Mitchell, and Folsom.

Dr. C. L. Dana then read a paper entitled "A Study of the Sensory Disturbances in Hysteria," which was discussed by Dr. Mitchell.

Dr. Sinkler then read a paper on "Recent Observations in the Etiology and Treatment of Migraine," discussion of which was postponed until the afternoon session.

Adjourned at 1.05 P. M.

Afternoon Session.

President Busey called the Association to order at 2.35 P. M.

Dr. Ernst exhibited a specimen of lepra bacillus, and presented a communication on the presence of bacillus tuberculosis in milk, supplemental to his paper presented at the last annual meeting, and, finally, made a preliminary report of Dr. Stephen C. Martin's work of isolating the *contagium vivum* from vaccine virus.

Dr. Trudeau next exhibited some cultivations of the bacillus tuberculosis.

On motion of Dr. Mitchell it was resolved, That the Association of American Physicians presents its congratulations and thanks to Dr. Martin for the interesting and hopeful research reported to it by Dr. Ernst, and expresses the hearty wish that it be carried to a successful conclusion.

Dr. Prentiss next exhibited a case of slow pulse and also a case of purpura hemorrhagica rheumatica, which cases were discussed by Drs. Mitchell, Graham, Atkinson, Jacobi, and Prentiss.

Dr. Sinkler's paper was then taken up and discussed by Dr. Graham.

Dr. Fitz then read "A Report of a Case of Acute Pancreatitis," which was followed by "A Report of a Case of Acute Diphtheritic Colitis with Peri-pancreatic Fat-necrosis," by Dr. Welch, and the latter paper was discussed by Dr. Fitz.

Dr. Shakespeare then read a paper on "What Can and Should be Done to Limit the Prevalence of Tuberculosis in Man?"—which was discussed by Drs. Trudeau, Johnson, Ernst, Bridge, and Shakespeare.

Adjourned at 5.30 P. M.

MAY 15TH.—MORNING SESSION.

President Busey called the Association to order at 10.20 A. M.

The Treasurer made his report, which was received and referred to Drs. Draper and Baumgarten as an Auditing Committee.

Dr. Kinnicutt, as referee, opened the discussion on "The Methods of Diagnosis in Diseases of the Stomach."

In the absence of the co-referee, Dr. Shattuck, his paper was read by the Secretary, and the discussion was continued by Drs. Peabody, Forchheimer, Welch, Roosevelt, and Kinnicutt.

In the absence of Dr. Smith, his paper on the "Etiology of Pleurisy, especially in its Relation to Tuberculosis," was read by title.

Dr. Graham made a report of two cases of acromegaly, which was discussed by Dr. Starr.

On motion of Dr. Fitz it was resolved that the President be authorized to furnish certificates as delegates to those members of the Association who desire to attend the International Medical Congress at Berlin.

On motion of Dr. Draper it was resolved that the Secretary be instructed to convey to the Surgeon-General the thanks of the Association for his kindness in furnishing it with a room for its meetings.

The Auditing Committee reported that they had examined the account of the Treasurer and found it to be correct.

The following officers, nominated by the Councillors, were unanimously elected:

President—William Pepper.

Vice-President—Henry M. Lyman.

Recorder—I. Minis Hays.

Secretary—Henry Hun.

Treasurer—W. W. Johnston.

The following gentlemen, whose nominations were approved by the Council, were elected members:

Drs. Hobart A. Hare and J. P. Crozer Griffith, of Philadelphia; Drs. T. S. Latimer and H. M. Hurd, of Baltimore; Drs. C. Denison and S. A. Fisk, of Denver; Dr. C. S. Bond, of Richmond, Ind.; Dr. R. L. MacDonnell, of Montreal, Canada; and to Honorary Membership, Dr. Fordyce Barker, of New York.

Adjourned at 12.15 P. M.

HENRY HUN, *Secretary.*

SOME POINTS IN THE NATURAL HISTORY OF ENTERIC OR TYPHOID FEVER.

By JAMES E. REEVES, M.D.,
OF CHATTANOOGA, TENN.

From the remotest period down to the present time medical history gives no era when the world was entirely exempt from epidemic contagious fevers. Indeed, the universality of their infliction—their reproduction an infinite number of times and under an infinite variety of local conditions—sometimes prevailing in a particular neighborhood and only attacking a few individuals; at others spreading amongst vast multitudes and cutting down with a wide swath of death and desolation all ages and ranks of the population; yet, with whatever scope of smiting visitation, whether epidemic and ravaging a continent or confined to a city, a hamlet, or rural hut, they have never been wholly extinct; but their specific germs have lurked a fatal spark among the embers of society, ready to burst forth into a consuming blaze at every favorable breeze.

An Unstable Nomenclature.—Much of the confusion which has existed concerning the natural history of some of the more prevalent specific pyrexial diseases has, no doubt, been due to an unstable, and perhaps sometimes unmeaning nomenclature. This reflection, I think, is allowable in connection with the study of the disease under consideration; for even since the beginning of the present century at least a dozen different names have been proposed and employed by systematic authorities.

Following the example furnished by the late Professor George B. Wood, M.D., whose memory I shall always cherish, because of his goodness to a poor young man in the backwoods of Virginia, I have chosen the name employed in the caption, and in recording my observations shall exclude all unnecessary reference to both the literary

history and the etiology of the disease, thus restricting the measure of my self-imposed undertaking to a sincere endeavor to present for your consideration and approval a true or natural picture of enteric or typhoid fever as I have seen and studied it by the light of nearly forty years' experience—alike in sparsely settled country districts, in village, town, and city; and when influenced neither by crowd-poison, medical treatment, nor the impress of particular idiosyncrasy.

The name *typhoid fever* was given by Louis in ignorance of a part of the ground, for he did not know, certainly, that another fever existed which ought to be called *typhus*, and in this uncertainty he named the disease *typhoid*, so as to convey, at any rate, the idea of analogy, if not identity, with what had previously been called typhus. In a letter addressed to me, in 1859, Professor Wood said: "Now that the fact is recognized that these"—typhus and typhoid—"are two wholly distinct diseases, it seems to me quite unphilosophical to give to one of them a name from its supposed resemblance to the other. We say *varioloid* very properly because the affection so called is a modification of *variola*, and depends on the same cause; but the typhoid is not a modification of typhus, and depends on a different cause. It is true that in some cases, and in certain symptoms, it is like typhus; but scarcely more than it is, in other instances, like miasmatic remittent. It is certainly, I think, not more like typhus than roseola is like *scarlatina;* and yet we do not call the former *scarlatinoid*, or the latter *roseoloid*. Beside, the word typhoid has before been applied to a state occurring in numerous febrile diseases resembling typhus; as a typhoid state of pneumonia, dysentery, bilious fever, etc., and its application to any one disease at present may lead to confusion, as, in fact, it often has led in the mind of the learner. The name enteric fever is unobjectionable, and will, I think, ultimately triumph, because it merely indicates that the intestinal affection is characteristic, as in the case of smallpox, which we name from the eruption."

Not a New Disease.—Belief in the spontaneous origin of the disease has greatly retarded the discovery and general diffusion of correct knowledge on the subject; for, by parity of reasoning, such faith is without substantial support when the question is studied with the life-history of the human race; while, on the other hand, the doctrine of permanency of original types of contagious diseases has, probably, as remote antiquity for its origin, as stable foundation for all future time, and

as many unanswerable arguments in its support as the doctrine of the permanency of the different types of the human race—each type with its entirely different capacities, physical and mental, and known to have existed for at least five thousand years, and to have been as much so at that long-ago period as at this moment; retaining its specific characteristics through every variety of circumstance, climate, and condition.

Now and then fortuitous circumstances may, indeed, have produced a hybrid; but such product could no more have given origin to a distinct and permanent species than a European can be changed into a Bushman, or, *vice versa*, from naked black Hottentot savages into Bacons and Shakespeares.

And thus, no doubt, through a long line of successive ages has enteric fever descended without change of its original or essential characteristics, and will continue its presence to afflict humanity in all situations where are congregated favorable atmospheric, terrene, civic, and domiciliary conditions for the growth and multiplication of its specific germ or contagium, so long as disease and death shall compass the earth.

Epidemic Visitation.—Epidemics do not prevail at all times; therefore, some special predisposing circumstance or influence must be present and lend its aid to produce widespread visitation. The contagion of measles, scarlet fever, diphtheria, whooping-cough, or smallpox, like that of enteric or typhoid fever, is probably never wholly extinct in any country where these diseases have once prevailed; yet their epidemic prevalence only occurs in uncertain cycles or during particular seasons. To determine why epidemics rise and spread in some years so much more generally or widely than in others, is one of the most interesting points in medical physics; but, unfortunately, no satisfactory answer has yet been given to this curious and most interesting question, and, while the phenomenon cannot be explained, we must be content in our ignorance to refer it to the influence of what was called by Sydenham and the older physicians, "The medical constitution of the air." Precisely the same uncertainty occurs with the crops; this year luxuriant growth and an abundant harvest; next year a general or partial failure; but the *seed*, under all modifying or controlling influences of growth and multiplication, remains the same in quality. In other words, the crop may vary as to the *quantity* or

abundance of the harvest, but the specific *quality* is unchanged under all conditions.

Again, some soils are absolutely sterile for the growth of certain grains. For example, Indian corn does not grow to perfection in Ireland, while wheat, rye, oats, and barley in that country come to maturity and yield the husbandman a successful harvest; and the same is true of certain localities concerning the prevalence of enteric or typhoid fever, also of diphtheria and scarlet fever, as I shall show on a subsequent page.

Definitive Elements.—Enteric or typhoid fever may be formulated thus: An acute contagious febrile affection derived from an antecedent case of the disease and depending for its communication from person to person upon the presence of a specific microörganism of, believed to be, well-known morphological character—a *bacillus*—which germ is capable of cultivation outside of the human body; prevailing less frequently in tropical and sub-tropical countries than in the temperate zone; both endemic and epidemic in its visitations; attacking, preferably, young persons and those in middle life; of long duration, but varying from ten to forty days *beyond* or in addition to the *prodromic* period which may cover from one to ten or even twelve days; if not profuse diarrhœa, a relaxed state of the bowels, and at all stages a marked susceptibility to the action of laxative medicine, with "pea-soup," or "ochre-colored" stools; intestinal hemorrhage in many cases, and in fewer instances perforation of the bowels; epistaxis, either slight or profuse; tympanites, with gurgling produced by pressure over the ileo-cæcal valve; dulness of hearing with mental hebetude, or morbid vigilance; headache disappearing during the second week; delirium, either mild or violent, with *subsultus tendinum;* rose-colored lenticular eruption during the second week, coming out in crops and disappearing in the same order; fever-curve showing steady elevation of from one to two degrees in the evening with a fall of the same measure in the morning for the first four or six days, and unlike that of any other acute febrile or inflammatory disorder; cough and bronchial râles; singular prostration of muscular strength; early heart failure and a dicrotous pulse; the tongue soon becoming dry and showing, in grave cases, a brown stripe in the middle, with the accumulation of *sordes* on the teeth and gums; in fatal cases, death during the third or fourth week; constant and

characteristic lesion of the small intestine involving Peyer's glands; enlargement of the spleen; convalescence tedious; true relapses not infrequent; and affording as perfect immunity from subsequent attack as in the case of scarlet fever or smallpox.

Partial History in Virginia.—Soon after my start in medical practice in Northwest Virginia—now West Virginia—in 1850, I encountered the most virulent and widespread visitation of enteric or typhoid fever it has been my fortune to witness, and I have read nothing in medical history of its epidemic prevalence and fatality, in this country, at least, which exceeded the ravages to which I allude. In thirteen adjoining counties the disease pursued its fearful march without respect of age, sex, or domiciliary condition. In fact there was scarcely a neighborhood in all that elevated or mountain country which was not visited and scourged. The towns and villages did not suffer more severely than rural districts, and so varied was the gravity, in some cases, so mild or so slow and tedious; in others, so malignant and rapidly fatal, that the disease received very different names to characterize its severity and fatality. In some neighborhoods it was given the name of a well-known family that had been swept off by it; in others, in which from the beginning the cases were malignant, and quickly fatal, it was called the *Black Tongue!* and there being such firm and general belief in its contagious character, sufficient nurses for the sick could not always be secured, and from that cause, in many instances, the most dire results followed.

I remember some poor families who were thus afflicted and distressed, and when death came to their households they were compelled to put away with their own hands, for want of neighborly assistance, the bodies of their dead.

In other instances, because of the frightful mortality even when the cases were under the treatment of the most experienced physicians, faith in the power of medicines to do good in the disease was lost in many families, and thus the *vis medicatrix naturæ* was left alone to battle with the pestilence. In these examples it appeared, at first thought, that they did equally as well as those who had the diligent service of their physicians, hence the opinion prevailed in some localities that patients were better off without medicine: in plainer words, "without a doctor." Again and again had I the opportunity during that epidemic of observing the march of symptoms where

nothing was done for the sick other than the administration of warm teas, and the allowance of plenty of cool water and a simple diet of milk and bread. But such management was not invariably successful in conducting the patient to safe convalescence; indeed, it was really less successful, but the deaths, several in number, were charged to the will of God.

It was by such impressive lessons at the very threshold of my experience in practice that I formed my acquaintance with the disease, and became assured of the value of the great truth that "violence in therapeutics is never justifiable when moderation is adequate to the same good end." And this is the foundation on which, year after year since that time, I have laid my observations and builded the confidence which brings me before you to-day with a fresh offering on some points on the natural history of enteric or typhoid fever.

Forms of the Disease.—In my studies I have been naturally led to recognize *three forms:* the simple or mild; the intermediate; and the malignant, including the fatal—rather than a division of stages by weeks; and I think this arrangement fully warranted by the clinical history. It may be extremely difficult in some instances to say just where the distinguishing or dividing line should be drawn, so very gradual is the transition from one form into the other; but in the large majority of cases there is but little difficulty in recognizing it.

Incubation.—The period of incubation, or the length of time between the date of exposure and inception of the specific poison, and the first manifestations of the disease, is as yet an unknown quantity, because the precise date of infection can but rarely be ascertained; but its duration is, probably, in most cases about two weeks. Instances, however, have been given where the disease followed immediately, or within a day or two, after exposure.

Mode of Access.—The initial symptoms or mode of access may vary considerably. In a few instances, there are no premonitions; in others the formative period may be extended from two to six, eight, ten, or even twelve days; and these differences may occur without meaning or foreshadowing the exact type of the fully developed disease.

As showing the suddenness of attack in some instances, I give the case of Mr. W., a young lawyer, who while engaged in defending a client indicted for a felony, and when reaching the climax of his

argument was so completely dazed for the space of a half minute or more, that he stood motionless, speechless, staring at the judge. His long pause soon attracted general attention in the courtroom; but just at the moment of greatest surprise at his conduct, he came to himself, connected his speech with the word at which he had left off, and went on to its conclusion without further embarrassment either of thought or speech. When he went into the courthouse that morning he was in his usual good health; when he left it that afternoon to go to his home, not far off, he felt physically unwell, and mentally oppressed with the weight of some impending dreadful calamity. Feeling confident he was about to become dangerously sick he at once prepared his will and then went to bed. Within the first week after his illness, he became delirious, and rapidly passed into the gravest form of the disease from which, six weeks later, he barely escaped with his life.

Entirely the opposite of the case above given, in mode of access, was that of Mr. T. G., aged thirty-six, clerk of the county court, who drooped for two weeks before finding himself unable to pursue his accustomed business employment, and the necessity of confining himself to his house. The next three weeks he rose every morning, dressed himself as usual, and spent the day in reading, writing, or other light engagement to while away the weary time; resting and napping on the sofa, as his inclination prompted. At the expiration of the period just named, I was called hastily early one morning to learn that a short while before my arrival he had been suddenly seized with severe pain in the abdomen while washing his hands and face, just after rising from his bed and partially dressing himself. His countenance was pinched, and pale as death, and he died within the next thirty hours from perforation of the bowels.

Between these wide extremes of mode of attack the *prodromic* period may be as varied as there are days in the limit; but, as a rule, the disease is fully developed by the fourth or fifth day from the time the first feeling of *malaise* is experienced. This declaration is based on the observation of one hundred and thirty consecutive cases in which I endeavored carefully to ascertain this and other not less important points concerning the disease. Headache and backache—accompanied frequently with slight nausea—start the clinical history. The pain in the head is usually referred to the occiput, but in many cases the head aches all over; while backache extends from the root

of the neck to the coccyx. There is a feeling of vertigo, and both mental and bodily languor are plainly manifested. The appetite is impaired, the tongue furred, and the mouth pasty, with offensive breath, and unnatural thirst. A shivery feeling now and then comes over the surface, alternating with flushes of heat; the pulse quickens, usually ninety to one hundred in the minute; sleep is interrupted by an evening exacerbation of febrile excitement, and the patient awakes without feeling refreshed; the urine is somewhat scanty and high-colored; and the bowels either simply relaxed or the condition that of free diarrhœa, in either case, accompanied with a sensation of increasing fulness of the stomach.

Hour after hour, or day after day, all these ill feelings become more pronounced and distressing, when, at last, a sensation of chilliness more severe and prolonged is felt, with an aggravation of the headache and backache, and the patient now willingly goes to bed to take on and pass through one or the other of the forms of the disease classified on a preceding page.

The Mild Form.—Sixty-four of the one hundred and thirty cases above referred to were of the mild form, a majority of which terminated in convalescence between the thirteenth and eighteenth day—the range for the whole number being from nine to twenty-five days.

In this class of cases, following the exacerbation of symptoms which completes the measure of the *formative stage* and ushers in the fully developed disease, the pulse becomes more frequent, say from ninety to one hundred and five in the minute; increased heat of skin, the rise of temperature marking a gradual increase of from one to two degrees higher in the evening than in the morning for the space of four or six days at the end of which time the range is between 101° and 104° Fahr., in the evening, with a corresponding fall in the morning of from one to two degrees. Along with the steady rise of temperature, the respiration becomes more hurried, and slight cough sets in and is accompanied with quite free expectoration toward the close of the fever. The headache continues from five to ten days, and when it ceases is usually lost in the obtuseness or drowsiness of manner which is such a common trait during the second week of the disease. There is throbbing of the temples, the face flushed, and toward evening when the fever is highest purple-colored spots come out on the cheeks—first upon one cheek and then on the other; or the nose may present the

same livid color and swollen appearance. The tongue may be coated so thinly as scarcely to be set down as coated, or covered with a dense white or yellowish fur, thickest at the middle and occasionally dotted with aphthous-looking specks; and is moist when the patient is not sleeping. At its tip and edge the coating loses itself, leaving the mucous membrane clean, bright and shining. In many cases the fur disappears in large patches, giving the appearance of loss of the mucous membrane at such places of its surface. If *malaria* complicate the case, the tongue is broad, flabby, and presents a creamy coating; otherwise it is not changed in size, does not assume the pointed shape, or show the brown stripe down its middle, so common a condition in the graver forms of the disease; and does not tremble when protruded. The abdomen is tumid, tender, and gives a gurgling sound from the right iliac fossa when deep pressure is made over this region; wandering pains in the stomach and bowels may be complained of, accompanied with nausea and occasionally free vomiting; loss of appetite, if not entire disgust of food; thirst excessive, the patient calling for water every few minutes; the bowels inclined to looseness and sometimes free diarrhœa; the discharges of the "pea-soup" variety, flocculent, and of peculiar and very offensive odor. The secretion of urine is diminished, highly colored, and turbid. Sleep is not refreshing, and the nights and sleeping hours are disturbed by unpleasant dreams. A very common mental condition during sleep is the impression that the body is severed in pieces, and much worry is felt in the unsuccessful effort to reunite the fragments; or, if not this trouble and distress, some other imaginary accident befalls the patient, and in his effort to extricate himself, wakes suddenly and manifests confusion as to his identity and whereabouts.

Dulness of hearing is one of the most unvarying conditions in the disease. Epistaxis is also exceedingly common, and several times repeated during the first two weeks. The same is true of the frequency of the rose-colored lenticular eruption. Indeed, I have but seldom failed to find it at some stage of the disease, generally between the seventh and fifteenth day. In my experience, it has been the rule to discover the greatest patches of this eruption in cases which were least troubled with profuse diarrhœa. In other words, it has seemed to me that the cases showing the least extent of intestinal alteration are the ones in which the eruption is most extensive.

Sometimes I have seen the patient spotted from head to foot; in other instances the eruption was principally confined to the chest, abdomen, and inner part of the thighs; or showed itself on the fingers and toes, while but few spots could be found on the trunk. Again, I have frequently seen it thickly set on the back, and am inclined to believe that it occupies this part of the surface oftener than is generally supposed, for the reason that it is not so conveniently sought for in this region as upon the abdomen, chest, and extremities. Of the vesicular eruption called *sudamina*, I have but this to say: I have seen it in but very few instances, all of which were in cases of the form of the disease next to be described, and preceded by profuse perspiration. Finally, there are but few cases of enteric or typhoid fever in which enlargement of the spleen may not be discovered.

Amelioration of these symptoms, shown by diminished temperature, skin relaxed and gently perspiring, refreshing sleep, cleaning tongue, relish of food and diminished thirst, stools less frequent and the discharges more consistent, increased secretion of urine, soft and flattened abdomen, and brighter countenance but with marked shrinking of the features, is the pleasing warrant of Nature's triumph, and that convalescence has been reached. Yet, such a happy termination may be suddenly thwarted by an aggravation of all the symptoms and the patient placed in greater danger than before; or he may suffer from a true relapse—the cause of which it may be impossible to tell—and go over again and through greater dangers the road he has just travelled. And thus the case may run a mild course throughout or take on at the beginning of the illness the graver character of symptoms I have distinguished as belonging to the intermediate form.

The Intermediate Form.—In this class there are but few additional symptoms to be recited that have not already been included in the description of the mild form. Thirty-two of the one hundred and thirty cases before referred to presented either at the beginning of the attack or during the second week the exasperation of symptoms characteristic of this form. With the same variable period of incubation and difference in mode of access, the febrile exacerbations are more prolonged and followed less frequently by moisture of the skin. The temperature rises gradually to 104½° or 105° in the evening and makes a fall of not more than a degree or two in the morning. In other words, the high temperature is more constant at all hours of

the day and covers a longer period. The pulse also is much accelerated, and soon becomes dicrotous because of the weak heart; the tongue takes on a thicker and darker coating except at the tip and edge where it is red and shining, and it presents also a dark-brown stripe down the middle. Its surface is dry, concave, with thin edge, and apparently contracted, resembling somewhat in form the bowl of a tablespoon with its point bluntly rounded, and cracked. When told to show his tongue the patient is slow in comprehending the request, and the effort is a trembling one, showing blunted sensibility and grave systemic involvement. At the same time *sordes* begin to form on the teeth, gums and lips, and the angles of the mouth are cracked and bleeding. Somnolency becomes more and more profound as the case advances toward the crisis (except in the fewer instances where is present morbid vigilance instead); the countenance dusky, and the mind and senses greatly blunted. In many cases the dulness of hearing is so extreme that it is with difficulty—loud calls and hard shaking—the patient can be aroused and his attention secured.

Wandering of the mind, at first only noticeable at night—especially during the second week of the fever—is now present at all times, both day and night, in the majority of cases. He sees the bedposts transformed into disagreeable personages who have taken their position at the foot of his bed to annoy and insult him by their grotesque performances; and under this provocation he starts up in anger to drive them from his presence, when the physical effort, thus put forth, dispels the illusion and he falls back upon his pillow wondering how it was possible he could have been so much mistaken. Soon, however, his Quixotic sally is repeated, and in that way he goes on day by day and night after night, sleeping, dreaming and wandering, except when his attention is rationally engaged.

In this condition, notwithstanding the continued high temperature, dry and parched tongue, the patient does not ask for cooling drinks; in fact, he has now become to a great degree insensible to his natural wants, for when his bowels are moved the disposition to empty them is not felt until just the few moments before the discharge takes place, and in the hurry to get him on the bed-pan, the sheets and other bedclothing are frequently soiled. The same obtuseness permits the bladder to become, sometimes, greatly distended before the desire wakes or arouses the patient to the necessity of emptying it. Diar-

rhœa, either mild or profuse, is always present—I mean liquid stools—and the frequency of the discharge from the bowels governs, usually, the extent of meteorism. In other words, if diarrhœa is active or profuse, there will be in the same proportion more extensive tympany and abdominal tenderness. In many cases the diarrhœa does not exceed more than two or three stools in the course of twenty-four hours; but it is more common for their number to reach a half-dozen in that time. As a rule, the stools are darker and far more offensive than in the mild form of the disease, and they are often attended with sharp pain about the umbilicus. Now and then they are bloody; but the intestinal hemorrhage is not so extensive as in cases of the malignant form.

The urine, as already stated, is passed in less quantities, deeply colored, and often voided with much difficulty. Epistaxis may be so frequent and profuse as to put the patient in great jeopardy. The diminished number of rose-colored spots in this form of the disease has already been stated; and it is also unnecessary to repeat what I have said concerning the presence of *sudamina*. Great prostration of muscular strength is an invariable condition, and, in a vast majority of cases, *subsultus tendinum* is present. It is in this form also that we find, very often, troublesome cough and sputa streaked with blood.

And thus the intermediate form may go on undetermined for a few days only or for many days, flattering the physician at one time with the approach of convalescence, at another time exciting his deepest anxiety. At the close of the second week, or about the beginning of the third week, it is not at all uncommon to find that a great improvement in the symptoms has taken place quite suddenly, so much so that the attending physician may find, since his visit the day before, that the tongue has become moist and has cleaned a little, the countenance brighter, and in every other particular a correspondingly happy change; when, as suddenly and without seeming cause, even a worse state of the case than at any previous time has taken place. This unfavorable turn of the symptoms may continue but a few days before convalescence is reached, or it may send the patient into the gravest form of the disease, now to be described.

The Malignant Form, including the Fatal.—Enteric or typhoid fever may assume the malignant form in the beginning of the attack; but this is not generally the way of the clinical history, for in the

majority of cases it is preceded, for a longer or shorter period, by one or the other of the milder forms. For example, of the one hundred and thirty consecutive cases to which I have before referred, and which in part of my experience furnish the basis for my positive opinions on this and other equally important points, twenty-five cases passed through the milder gradations, with variable duration, before reaching this form. So, in order to present a continuous and faithful description of the disease from the moment of development to its termination, it is necessary that I go back to the point at which I completed the description of the *intermediate form*, and then proceed with the graver assemblage of symptoms to the end, either in recovery or death!

With the exasperation of symptoms which marks this form of the disease, the progress is then usually rapid to the final termination. Prostration of strength is an ever-present condition. The patient is now but rarely found on his side, for he has not sufficient strength to maintain that position and must lie on his back, with a disposition to slip down in the bed. The pulse is frequent and feeble; constant twitchings of the tendons of the wrists, and sometimes the whole body is in constant tremor from muscular twitchings, to such extent in some cases that even the bed-clothing is in constant wave-like movement, or if one sit on the bed-rail a trembling sensation is distinctly perceived. The voice becomes distinctly changed, so feeble in some cases that it can scarcely be raised above a whisper; in others the articulation is so imperfect that all attempts at conversation amount to but little more than a trembling jargon. The tongue takes on a thicker and black coating, and all efforts to protrude it are slow, trembling, and uncertain of success, and when shown, if the patient is not told to take it in he will likely leave it exposed. With this state of the tongue and general insensibility, deglutition is often so much interfered with that food and drinks are taken with much difficulty.

The temperature of the surface may be uniform or unequal; the head and trunk hot and dry while the extremities are cool, or the face and hands sweat freely while other parts of the body are dry and husky. Along with this grave condition cough is more or less present in every case, accompanied at one time with free expectoration, at another nothing but frothy, sticky mucus is expelled; loud *râles* are heard, breathing becomes hurried and occasionally irregular or jerk-

ing. The temperature line shows but little variation between the extreme of evening rise and morning fall; is fitful at all times, and of undulatory character. The condition of the abdomen and state of the bowels are also fair measures of the serious character of the case; the tympany makes a convex surface from the ensiform cartilage to the pubes, and when pressure is made the abdominal wall seems thin, hard, and resisting "as though made of pasteboard," and scattered here and there over its surface a few *rose-colored* spots.

The stools are very offensive, of dark color, and so sticky that the bed-pan or chamber-pot is emptied and cleansed with difficulty. In color and consistence the discharges resemble dark-brown paint; but when slight hemorrhage takes place and the blood is retained for a few hours, the discharges are black and tarry and exhale a most sickening odor. It is in this form of the disease always that the most frequent and profuse hemorrhages take place. In many cases the blood discharged from the bowels is red and but little changed; in others it is dark and disintegrated. Several times I have seen the usual sized chamber-pot half filled at a single sitting, and in one case the patient fainted and fell from his chair to the floor, and was put back in bed almost lifeless. In this case hemorrhage succeeded hemorrhage until the eighth day from the first bloody discharge, when he died. Eighteen of thirty-four cases, above referred to, suffered hemorrhage from the bowels, and of these, eight were fatal: In this form of the disease, I have seen the patient bleed from the nose, from the gums, from the bowels, from the bladder, and from the skin—*petechia.*

The urinary secretion shows not less alteration of character. It is scanty in quantity and in color dark as lye, and when there is hemorrhage from the bladder small particles of coagulated blood are deposited at the bottom of the vessel; or the urine may be retained, either with or without admixture of blood, requiring repeated use of the catheter. Delirium, so common in this form of the disease, may be either mild, violent, or low and muttering; the patient picks at the bed-clothing, or reaches out his hand to catch some imaginary object. In a few instances I have seen the delirium strictly monomaniacal. Involuntary discharges both of urine and feces frequently take place and then there arises from the patient's body a most nauseating odor. In addition to the above described conditions, the skin may break on parts most exposed to pressure, as the sacrum, hips, and

shoulders, and be followed by sloughing,—all these showing vitiated constitutional powers.

Finally, when the case is to end in recovery there is relaxation and improvement of all the symptoms, and the patient passes, sometimes quickly and at other times slowly, through convalescence to health. But if death is to be the end of the case, it may come in different ways, according to the lead of symptoms. If by coma—the most frequent mode of death in enteric or typhoid fever—the low muttering delirium from which the patient could at first be aroused, gradually becomes more and more profound until complete insensibility is reached; the jaw falls by its own weight and the mouth is open, the lips tremble, the muscles of the face twitch, the eyelids are half closed, the balls upturned and roll from canthus to canthus, petechiæ and vibices make their appearance on various parts of the surface, the parotids swell and may suppurate, the pulse beats so rapidly it can scarcely be counted, the breathing is rapid, and automatic efforts are made to expel the secretions which choke the lungs and rattle in the throat, a cold, clammy sweat breaks out, the extremities become cold, there is a final struggle and Death has won the mortal victory! In fewer instances the fatal result is reached by almost imperceptible degrees; from first to last the mind remains unclouded, and the patient passes away without a struggle.

Thus I have completed the description of a case either *mild* throughout, or having been mild for a longer or shorter time, passed into the *intermediate* form, then either convalescing or passing into the *malignant* form. Of the thirty-four cases of this form, nine assumed this character at the beginning of the fully developed disease, six of whom complained of feeling out of health for three days previous to taking their beds; the remaining three retired as usual and in good health to awake next morning with headache, sick stomach, and vomiting, a chill, and then fever. In all of these cases the mind and senses were quickly involved, as shown by aversion to light and sound, morbid vigilance, confusion of mind, followed as early as the second or third day by active delirium, which continued until lost in *coma*. After such beginning, the case proceeds as above described to its final termination.

Supplemental and Emphasis.—In presenting the foregoing description of the symptomatology, sufficient emphasis of certain conditions

seemed impossible without interrupting and confusing the connected clinical history of each phase of the disease; hence, I shall now endeavor to cover the deficiencies to which I refer.

Headache is so constant in its presence, during the first week of the disease, that it was found in 127 of 128 patients who were of sufficient age to express suffering. Sometimes it is exceedingly severe, assuming a neuralgic character, and it may continue through the whole course of the disease.

The state of the mind and senses varies with the different forms of the disease; from a blank, dejected expression of countenance, listlessness of manner and simple wandering of mind to the wildest delirium. A very common impression with the patient is that he is away from home and exposed to personal injury; thus influenced, he jumps out of bed if not restrained, and makes for the door. Delirium is equally common in children, but of milder form than in adults. Of 23 cases of the malignant form of the disease terminating in recovery it was present in 21; and of 11 fatal cases it was distinctly marked in 10, the exceptional case passing to death without a cloud to obscure its approach. Sometimes it is of long duration, continuing for three weeks, and it may present any grade or character. In other cases there may be a sudden outbreak of mental disturbance during the period of convalescence. In one such case after the patient had proceeded so far on the road to health as to be able to go out of doors for several days, he was suddenly overcome with illusions which continued for a few days and then as suddenly disappeared, leaving no impress or mark behind.

Dulness of hearing is so complete in some cases that in order to arouse the patient he must be spoken to at the highest pitch of the voice. In a few instances the reverse of dulness exists, and the sense may be so acute that the slightest noise attracts attention, even the light step of the nurse across the floor, and unless there is perfect stillness in the room the patient is painfully discomforted.

Prostration of muscular strength is such a constant condition from the beginning to the close of the disease—such a fair measure in forming the prognosis—that its importance cannot be too strongly emphasized. Even during the formative stage, and before the patient takes his bed, his gait is tottering, and if he attempt any manual labor he is soon fatigued. In the *mild* form when he is placed in a chair in

order that his bed may be made up and aired, he soon tires and is impatient to lie down. In the *intermediate* form he must have assistance to hold up his head for a drink of water. In the *malignant* form there is utter prostration. An increase of the strength is the sure harbinger of convalescence. In the so-called "walking cases," loss of muscular strength is the most that is complained of by such patients.

Accidents and Sequelæ.—The most fearful of the accidental inflammations is peritonitis the result of perforation of the bowels, the symptoms peculiar to which are so well known that it is unnecessary here to describe them further than to say the accident occurs, usually, in the third or fourth week—in rare instances during convalescence—and is announced, in the majority of cases, by the sudden supervention of acute pain in the abdomen, accompanied with either a pronounced and severe chill or prolonged feeling of chilliness, with cool, clammy skin; acid eructations and vomiting; abdomen exquisitely tender on pressure; great prostration of muscular strength; rapid, feeble pulse; and after recovery from shock, abrupt rise in temperature.

I have seen in five instances all the symptoms which announce and follow perforation of the bowels, yet the patients recovered; so that I am led to believe the accident is not necessarily fatal. Four of the five cases to which I refer occurred in males, all over thirty years of age save one.

The safety of pregnancy seems to be but little influenced or endangered by the disease, for in all my experience I have not met with a half-dozen abortions or miscarriages which could be charged to its presence. And there is good ground, I think, for belief that the pregnant state affords some immunity from the disease.

Among the sequelæ, I have seen frequent examples of *swelled leg and thigh*, like that which is incidental to parturient women, and first noticeable during convalescence. In one such case the swelling came on before the patient was able to quit his bed for the whole day; and in another case, at the opposite extreme, it was not noticed until during the third week after recovery from the fever. In some cases, especially those developed early in convalescence, the swelling is accompanied with pain and tenderness; but ordinarily this accident gives no other cause of complaint than a feeling of stiffness and slight lameness. I have never seen a leg thus affected go back to its normal size and proportions and get well.

Special Memoranda. Season.—The autumnal and winter months, according to my observation, give the largest number of cases.

Age.—A large majority of cases occur between the ages of fifteen and thirty years; yet I have seen the disease in an infant of eighteen months—the mother sick at the time from the same cause; and, at the other extreme, repeatedly in subjects above sixty years of age.

Sex.—The disease more frequently attacks males than females.

Race.—The disease is probably less frequently met with in the negro than in the white race.

Immunity in Certain Localities.—In an early part of this paper, when referring to the wide diffusion of the disease, I mentioned the fact that in certain places it had but rarely been met with, and never witnessed in endemic or epidemic form. Chattanooga is one of these remarkably favored places where enteric or typhoid fever does not prevail, notwithstanding the city has a population of 60,000, and is embarrassed by very serious sanitary defects such as are common to all new and rapidly growing cities, namely, insufficient sewerage, many miles of unpaved streets and alleys, and thousands of open cesspools in the form of the old-fashioned privy vault.

Dr. P. D. Sims, a member of the State Board of Health who has been in active general practice for more than thirty years in this community and is a most competent observer, says that in all of his experience in Chattanooga he has seen but three or four cases of "typhoid fever." A like testimony is furnished by Dr. J. H. Van Deman, who, until the last three or four years, had been engaged in general practice since 1865, or from the close of the war, when he quit the army. The observations of Drs. Sims and Van Deman have been confirmed by a score or more of later witnesses who from time to time have come to the city to help occupy the professional field.

Chattanooga is situated in the great Moccasin Bend of the Tennessee River, at the foot of Lookout Mountain, with an altitude of 650 feet above sea level, and surrounded by high hills and mountain ranges that rise above the city from 600 to 1650 feet. The prevailing winds from March until December are either from the southwest—sweeping the top of Lookout Mountain, whose base is but three miles distant from the centre of the city—or from due west, when they come laden with freshness and health from the Raccoon Range, but four miles away, which is 1400 feet above the level of the city. In winter

the prevailing currents come from Walden's Ridge, six miles to the north, which chain walls the valley on that side. In the undulating cove in which Chattanooga stands, with her "seven hills," like the Eternal City, she is sheltered on all sides; possessing a stable, genial climate, blessed with good general health at all seasons, and adding every year thousands to her thriving, busy population.

The United States Signal Service report from Chattanooga Station for ten successive years shows the temperature to be as follows:

Mean Temperature.

For the Year.		For Winter. December, January, February.	For Summer. June, July, August.
1879.	60.7°	44.5°	76.4°
1880.	60.3°	45.7°	76.2°
1881.	60.8°	42.6°	79.0°
1882.	60.3°	44.7°	74.9°
1883.	60.8°	45.7°	76.9°
1884.	59.6°	42.0°	74.2°
1885.	57.7°	39.2°	76.4°
1886.	57.3°	36.6°	73.9°
1887.	60.1°	43.6°	76.2°
1888.	59.1°	43.2°	76.2°
1889.	60.4°	47.2°	75.1°

The total rainfall for the same period was as follows:

Total Rainfall.

For the Year.		For Winter.		For Summer.	
1879.	4.34 inches.	7.60 inches.		8.88 inches.	
1880.	5.66 "	4.23	"	3.61	"
1881.	5.8 "	6.57	"	3.43	"
1882.	5.16 "	8.20	"	4.71	"
1883.	4.16 "	5.49	"	3.21	"
1884.	5.15 "	7.16	"	5.51	"
1885.	4.72 "	5.13	"	5.00	"
1886.	4.88 "	4.38	"	4.88	"
1887.	4.26 "	6.07	"	3.54	"
1888.	4.57 "	4.57	"	3.57	"
1889.	4.11 "	3.62	"	3.75	"

During the last three winters, or since my residence in Chattanooga, I have not seen snow on the ground.

The Annual Death-rate.—In ordinary years the death-rate does not exceed twelve per thousand per annum, including both races;

and if the entire population were white, it would not, probably, exceed ten per thousand.

Conclusion.—Diphtheria is also an unknown disease in Chattanooga, and the same is nearly true of scarlet fever. No doubt that, now and then, examples of all three of these diseases have been met with, brought in the persons and baggage of visitors; but the seed, when imported, uniformly failed to multiply.

The miasmatic remittent fever met with in Chattanooga sometimes assumes a grave character, and then often presents the so-called "typhoid state;" but its clinical history in every essential particular is wholly unlike enteric or typhoid fever. Since taking up my residence in the city, I have had good opportunity to observe the changes in the blood in malarial cases, and am entirely convinced of the value of Laveran's researches in striking the difference between malaria and enteric or typhoid fever. And here I desire to acknowledge my indebtedness to our own countrymen, Councilman and Osler, for their distinguished labors in the same field. Again and again have I been able, easily and quickly, to clear up the diagnosis by a microscopical examination of the blood in cases of doubt; for, in the language of Dr. Osler, "the characteristic changes in malaria are as distinctly determined in the blood as are those of tuberculosis of the lungs in the sputa."

DISCUSSION.

Dr. J. C. Wilson: Dr. Reeves's paper is extremely interesting and valuable as bearing on the natural history of enteric fever. There is no great danger that our information regarding the natural history of acute diseases will be too great nor too exact. It is especially desirable that full accounts of the clinical course pursued by the acute infectious diseases, in different sections of the country, and under different conditions of living, should be presented to organizations of this kind. The comparison of facts and the recognition of differences thus becomes possible.

Dr. Reeves's paper confirms the evidence that the enteric fever, which he has had the opportunity of studying in thinly-settled sections of Virginia, is identical with that observed in more thickly-settled regions and in the centres of population. Enteric fever is unquestionably the same disease everywhere and under all circumstances. Different cases vary in intensity and in duration, just as different cases of other infections show marked variations. The recognition of this fact must, in the course of time, dispel the doubts in regard

to the nosology of certain fevers prevalent in newly-settled portions of our country.

There are one or two points in Dr. Reeves's paper that especially attracted my attention: First. The subject of the eruption. He describes the eruption as being occasionally present from head to foot. Are we to infer that he has frequently seen it upon the face? Most of the text-books on the subject say that the eruption of enteric fever does not appear upon the face. There are, however, undoubted instances where it occupies the face. I have seen four such cases, all in children or young adults.

I am much impressed by the statement Dr. Reeves has made in regard to cases presenting all the appearances of peritonitis from perforation, followed by recovery. The occasional sudden onset of symptoms of peritonitis which follow this favorable course is a matter of general observation. It appears to me that further clinical and pathological studies must be invoked to establish data for the early differential diagnosis between the local plastic peritonitis, tending to recovery, and general septic peritonitis, from which, in the present condition of medical science, recovery is impossible. If operative interference is ever to save life in perforation of the intestine in enteric fever, it must be done early.

The opinion has become very prevalent that the clinical course of enteric fever is but little modified by treatment. In so far as the expectant-symptomatic treatment goes, this view is conceded on all sides to be true. The claim of Brandt, of Stettin, that the treatment by the cold bath, in accordance with his method, does modify the clinical features of enteric fever to such an extent that the classical picture of the disease is not seen, is unquestionably true. I have now under observation in the German Hospital of Philadelphia a series of thirty-seven cases, which have been treated strictly by the cold-bath treatment, in accordance with the directions of Brandt, with the result that he claims—namely, a striking modification of the course of the disease, especially as regards the morbid phenomena due to the nervous system.

Dr. A. L. Loomis: The interesting report which Dr. Reeves has given us tallies very well with the history of the disease as it has been observed. The three forms into which he has divided this fever I have not recognized in practice.

The terms mild and severe, typical and non-typical, endemic and epidemic, have seemed to me sufficient to include all the varieties of typhoid fever. The intermediate fever described by the writer I have never separated and studied as a distinct type. The term seems to me somewhat confusing. Whenever typhoid fever becomes epidemic, as it occurred in Western Virginia, certain atmospheric and soil conditions must exist, which would modify the type and severity of any acute infectious disease which might happen to be prevailing at that time.

The writer rightly classes peritonitis as the most fearful accident of typhoid fever. But I was surprised at the statement that he had five recoveries after perforation of the intestines. I do not remember to have seen a single

recovery after there were unmistakable evidences of intestinal perforation. Recovery from a local peritonitis complicating typhoid fever is not unusual, but when the characteristic symptoms of intestinal perforation are present, in my experience a fatal issue soon follows.

I cannot conceive of recovery after the intestinal gases have escaped into the peritoneum.

Œdema of the lower extremities in the majority of instances I have found due to venous thrombosis. I have noted thirty-seven such occurrences with only three deaths.

Œdema of both extremities from general phlebitis of the superficial veins I have met in three instances, with two deaths and one recovery. Phlebitis of the left femoral vein, in an alcoholic subject over fifty years of age, was followed by gangrene of the foot.

I have the records of one case of thrombosis of the femoral artery with acute arteritis, occurring as a sequela of typhoid fever, accompanied by œdema of the extremity. My own experience leads me to the conclusion that in every case of œdema of the lower extremities occurring either as an accident or sequela of typhoid fever, it is important to determine the pathological changes which have led to it.

The cardiac softening (as Hayden terms it), which may be regarded as a part of the history of typhoid fever, is usually entirely recovered from, but those cases which are attended by a loud systolic murmur at the apex, with other signs of marked heart insufficiency, are liable during convalescence to two serious accidents—cardiac thrombosis and excessive cardiac dilatation. The danger of excessive dilatation from sudden heart strain during convalescence in those who give marked evidence of heart insufficiency is so imminent that the greatest care should be exercised during the early period of convalescence.

In one of my cases of old valvular disease, typhoid fever during the fourth week was complicated by acute ulcerative endocarditis, causing infarctions in the lungs, spleen, and kidneys.

Subacute gastritis is another accident which has occurred to me several times in the convalescence of typhoid fever from imprudence in diet during the early period of convalescence. I remember the case of one young woman who ate largely of ham and eggs during the fourth week of her fever, who died with the symptoms of acute gastritis.

Dr. Ross, at the meeting of the Association in 1888, presented an able paper upon the sequelæ of typhoid fever, and one to which little can be added in the line which he followed.

It has occurred to me, however, during the past year to meet with a peculiar condition occurring as the sequelæ of typhoid fever which I have not seen noticed except in the pamphlet which I hold in my hand. This is a paper presented to the Orthopedic Association by Dr. Virgil Gibney.

Four cases are given in that paper in which certain symptoms were present which were not accounted for by any change in the spinal column itself. One of these cases was a patient who was under my observation. He was a man,

eighteen or twenty years of age, sent to New York from Schenectady by his physician. He had typhoid fever in November and was thoroughly convalescent by the 1st of January. About the 20th of January he first experienced severe pain in his back. This pain became so severe and his inability to move so great, unable even to change his position in bed without crying out, that he was compelled to keep his bed. He was at first treated for lumbago; afterward, about the 1st of March, he came to New York. There was intense pain on the slightest motion of the spine and exquisite tenderness on pressure along the lumbar vertebra. I requested Dr. Gibney to see him with me in consultation. Dr. Gibney after examining the case suggested that it was one of the cases to which he had given the name of *typhoid spine*. I requested Dr. Gibney to take charge of the case. He applied the cautery over the points of tenderness and placed him in a spinal jacket. In the course of about two months the patient was entirely well.

DR. S. WEIR-MITCHELL: I want to say one word in regard to this last case cited by Dr. Loomis. I did not understand whether any local study of the temperature was made. I think if it had been he would have found a slight rise of temperature at the points of pain. I have seen three or four cases of this kind coming out of typhoid fever, once or twice out of prolonged malarial fevers. They had the characteristics of the case reported by Dr. Loomis, but in all of them I found a slight rise of temperature.

DR. H. A. JOHNSON: I want to state a fact which was a matter of personal observation many years ago in the locality in which I then lived; a region in which the diseases were almost all malarial. A starch factory was established for the purpose of making starch from potatoes, and the farmers around raised a large quantity of potatoes for this use. Some delay in regard to the machinery occurred and nearly a hundred thousand bushels of potatoes decayed. This was on the bank of a little stream. There broke out in the next month or two first, an epidemic of cerebro-spinal meningitis, which had never been known in that region; this was followed by typhoid fever. It was a new disease. The physicians had seen no cases of the fever before. From that time on it continued to prevail in that locality.

The point which I wish to bring before the Association is this, that in a region remote from typhoid fever there broke out an epidemic of typhoid fever immediately following an epidemic of cerebro-spinal meningitis, both preceded by the decay of a large quantity of potatoes in mass. The first cases occurred down the stream from the location of the starch factory. It raises in my mind, and always has, this point: whether non-pathogenic bacteriæ or bacilli may not be so modified by cultivation under peculiar circumstances as to be the efficient cause of diseases such as have been described. The decaying mass of potatoes was evidently a very fertile culture medium.

A case came under my observation bearing on the question of recovery after perforation of the peritoneum. A patient who was believed to be conva-

lescent had a sudden collapse. I saw him a very short time afterward and suspected perforation, and believed the case to be hopeless. I, however, placed the patient under treatment—ice to the ileo-cæcal region, stimulants, opium, etc.—and, to my surprise, he did not die, but developed a tumor in the ileo-cæcal region.

The patient gradually got well, and it has been a question whether it was a localized peritoneal inflammation from extension with the characteristics of perforation, or whether the man recovered from perforation.

DR. A. JACOBI: I certainly do not want to prolong the discussion, but would like to make a remark in reference to the introduction of a new name into our nomenclature—typhoid spine.

It may be one of two things. In the absence of temperature it may be neurosis, or it may be spondylitis. Mild forms of spondylitis are not so uncommon as they are believed to be. It appears to me that one of the two must have been the matter here, and that it is rather problematic to introduce a new name into our nomenclature, which indeed would not mean anything but that the exact diagnosis was uncertain.

DR. WM. PEPPER: I think the more strongly we set our face against new names the better. The reader of the paper has entered a mild protest against the name typhoid itself, but after all it is usage which must guide us in the use of terms. Of the multitude of names suggested for this disease, the word typhoid seems to be the only one universally acceptable. We should do nothing toward substituting another name for this until our advanced accuracy of diagnosis enables us to designate it by the true pathological factor which causes this specific disease.

One of the most interesting things in Dr. Reeves's paper was the statement of the extraordinary immunity of Chattanooga from typhoid fever. I trust we shall be favored with further reports from this locality. If general experience is reliable, it may be expected that ere long there will be there an outbreak of the fever. Refering to the term malignant typhoid, it seems to me we must recognize such a disease; since, although rarely in comparison with scarlatina, we do meet with cases where the intense virulence of the poison strikes the patient down at once, and he dies without time for the development of the disease. These cases are fortunately rare, but are to be met with and verified by post-mortem.

Dr. Wilson in his remarks as to the modification of the natural history of typhoid fever and other infectious diseases by therapeutic agents, introduces a large and very important subject. I think we ought to speak frankly on occasions like this, and I never like to lose an opportunity to testify as to my belief in the value of nitrate of silver as a factor in the treatment of typhoid fever. When I first began to use it and to teach its value in this disease, I was unaware that it had been recommended by the late Prof. John K. Mitchell. But for years I have now administered it, in hundreds of cases, and my correspondents in various parts of the country have reported to me the effects of

this treatment in large numbers of additional cases. It appears that, when it is given at the earliest possible moment after the nature of the disease is suspected, and is continued steadily until the close of the fourth week, it exerts usually a beneficial effect on the development of the intestinal lesion, and indirectly upon the general symptoms. Of course its use is compatible and should be combined with such other remedies as may be indicated in particular cases. Of course, also, it is essential that strict rest and suitable diet be insisted on. I shall not now enter upon detailed statistics, nor upon any discussion of its supposed mode of action, but must limit myself to this expression of my confirmed belief in the value of the internal administration of nitrate of silver in typhoid fever.

Dr. Reeves: I have but few words to add in closing the discussion. In answer to Dr. Wilson's question as to the frequency of the eruption on the face, my paper asserts in general terms that I have but rarely failed to find the eruption at some stage of the disease; and this is also the answer to Dr. Dana's inquiry.

To Dr. Loomis's criticism, I reply that I have specifically stated, after summarizing the symptoms of perforation of the bowels in fatal cases, that the same order and character of symptoms had been presented in cases in which recovery took place; therefore, I felt warranted in believing perforation of the bowel was not necessarily fatal. Of course, whether or not there was perforation in the cases referred to, no one can tell. The unerring history can only be found on the post-mortem table.

In reply to Dr. Johnson's remarks concerning the outbreak of cerebro-spinal meningitis followed by a visitation of typhoid fever, I have simply to say that I have witnessed the prevalence of both diseases at the same time in epidemic form. Again, because enteric or typhoid fever made its appearance in a community where before it had been unknown, is no proof that some person having the disease, but unrecognized as a carrier of contagion, may not have gone into the community and deposited the specific germ which started this epidemic. Until that question is settled, the history given by Dr. Johnson cannot be discussed. I have no more faith in the *de novo* origin of enteric or typhoid fever than I have in the spontaneous origin of smallpox. There must be an antecedent case to start an outbreak of the disease.

Referring to the remarks of Drs. Pepper, Mitchell, and Jacobi, I may make answer that there is nothing in the mere appearance of the soil in Chattanooga —both surface and substrata—that differs in the least from soil conditions in other places in Tennessee, Georgia, and Alabama, where, it is said, the disease more or less frequently prevails. The city water-supply is from the Tennessee River, and the intake to the waterworks is safely above the point of sewage and other contaminating inlets. But it should also be stated that the same immunity from enteric or typhoid fever existed when the inhabitants received a much less pure water-supply than at present—when well water, also river water taken from below sewage inlets, was used.

Finally, if it shall be your pleasure, gentlemen, at some future meeting of the Association, and when the spirit moves me, I shall be glad to follow this paper with one on the Diagnosis and Treatment of Enteric or Typhoid Fever. I confess now that I have but little faith in the curative effect of the cold-water bath, and should feel very grave responsibility in advising the plan to my patients. Its employment may, possibly, be successfully accomplished in hospitals, but in private practice it is impossible, I think; and, probably, would do more harm than good.

INFLAMMATIONS OF THE APPENDIX AND CÆCUM, AND THE DUTY OF THE PHYSICIAN REGARDING THEM.

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To anyone who reads the recent literature of this subject it will be apparent that it has received its chief study of late, not at the hands of general medical practitioners, who have the greatest opportunities of observing cases of these inflammations, but at the hands of surgeons—men who make surgery a specialty, and who see few cases, in their incipiency, of inflammation of the abdominal organs. The character and effects of these inflammations have been revealed to a large degree by post-mortem examinations, but it was left for modern surgery, with its antiseptic methods, to illuminate the subject of the treatment of many of their results, as well as their character and tendencies. In proportion to the number of cases falling under the observation and management of surgeons, the results in many ways have been profitable and encouraging. The surgeons have reached certain conclusions as to the character of these inflammations and what ought to be done for them, some of which are rather sweeping, and which encounter doubt and opposition on the part of many practitioners who give little or no attention to surgery. If the results attained by surgery could be put with the observations of large numbers of cases never requiring surgical interference, and which are never seen by workers in the field of surgery, we ought to have the safest guide for diagnosis and treatment.

It is certain that these diseases are frequently not recognized and are often mistreated, and that the notions of doctors generally regarding them need to be radically revised. An obstacle to the exact understanding of the whole subject is the reluctance practitioners manifest to reporting their unfortunate results and bad diagnoses.

One prevalent misconception regarding these inflammations is of their actual and relative frequency. Primary inflammation of the cæcum is rare, while that of the appendix is very common. Of all organs of the abdomen in males the appendix is most prone to dangerous inflammation. In females this tendency is exceeded by the pelvic organs only. Of 300 autopsies at random Toft reports that 36 per cent. revealed evidence of disease of the appendix. Such evidence doubtless consisted of adhesions of the organ to adjacent parts, and of various changes in its structure and condition that can only come through inflammation of some degree or character. Often these appearances are found in cases not known to have ever had abdominal inflammation. Four-fifths of all cases collected by Fitz were in males, showing a marked predisposition difficult to explain. While of all cases of appendicitis without extension only 20 per cent. were in females, 26 per cent. of cases of perityphlitis were in that sex. This does not prove, perhaps, that women are more prone to perityphlitis from inflammation of the appendix than men, but the increased percentage may be explained by the possibly greater tendency in them to pure cæcitis from foreign bodies—aggravated by constipation—since irritation within the cæcum sometimes leads to perforation and perityphlitis.

Dr. Ludvig Hektoen, of Chicago, a careful observer, has noted the condition of the appendix in 280 post-mortem examinations and found that in 42 cases (15 per cent.) there was evidence, in adhesions or otherwise, of previous peri-appendicitis from which the patient had recovered. Cases of existing inflammation were not counted. So in Chicago, among people who find their way into the County Hospital, fifteen in every hundred have had inflammation of the peritoneal surface of the appendix or the connective tissues posterior to it, or both, and have recovered. These figures are not inconsistent with the records of Toft, since he observed all diseases of the appendix both inside and out, while Hektoen only recorded cases showing inflammation of the outside of the appendix and which had recovered.

While primary inflammation of the cæcum is unusual, its secondary involvement from appendicitis and resulting perityphlitis or circumscribed peritonitis is doubtless common.

It must be extremely unusual for the cæcum to be primarily attacked except when irritated by foreign bodies within it. Even in such cases

it is rare for perforation to occur; this event usually comes from without, when a perityphlitic abscess opens into the cæcum.

The vast majority of cases of so-called typhlitis and perityphlitis begin as appendicitis, the tissues of and about the cæcum become inflamed secondarily, and usually as a result of perforation of the appendix. These two pathological conditions are often confounded; probably in most instances perityphlitis is the term to use. It is not the inflammation of the cæcum that is usually a menace to life, but that of the cellular tissue and peritoneum that so often leads to induration, and to abscess with its manifold dangers of spontaneous opening in perilous directions.

Probably failure of free evacuation of the appendix is the first step to the causation of its commonest inflammation. The moment its contents are unduly retained harmful decomposition begins, deposits of lime-salts occur upon retained fecal matter, if such is present, and enteroliths are formed which press too hard on the swollen and tense mucous membrane, which finally yields in the weakest spot and an ulcer is present. Only occasionally do similar consequences flow from foreign bodies in the appendix. In a considerable proportion of cases the ulcer occurs without the irritation of a hard body of any kind. The ulcer deepens by a persistence of its causes till it touches the internal surface of the covering peritoneum, or the cellular tissue if within the mesentery; then trifling peritonitis or cellulitis, or both occur, ending in a few days if the causative process stops. If, however, the ulcer deepens it perforates the appendix, a particle of its contents is extruded, when peritonitis or perityphlitis promptly ensues. In some instances the peritonitis is sharply localized and plastic exudation bars the irritating material from the general peritoneal cavity, and intra-peritoneal abscess forms; in others a general peritonitis occurs, and usually death in a few days. If the perforation is within the mesentery a cellular inflammation results and we have the common perityphlitis—usually not wholly independent of peritonitis—and in a large proportion of cases such induration as may be easily demonstrated, and within this pus.

Most cases in males of so-called "inflammation of the bowels," as characterized by many general practitioners, are simply instances of inflammation of the appendix or cæcum, with or without the involvement of the connective tissue about them, or the peritoneum, or both. It is

equally true that most cases of peritonitis in men and boys, and not a few in females also, are due solely to perforation and infection consecutive to these diseases. Until discovered post-mortem this cause is generally not surmised. I venture the assertion that most autopsies after peritonitis in the male sex reveal this etiology—and sections are made in only a meagre minority of such cases.

A case in point came under my observation in consultation about two years ago:

Mr. S., a large, robust man of forty-five years of age, had enjoyed apparent perfect health for many years. After attending a banquet he stood for half an hour, dressed in a thin suit and without an overcoat, in a cold and windy doorway waiting for his carriage. The next day he felt uncomfortable, later had some pain and slight tenderness in the abdomen, the next day fever and some vomiting. A consultation a day or two later discovered simply evidence of a mild general peritonitis; there was neither much fever, pain, tenderness, nor prostration, and the tenderness was localized in the lower zone of the abdomen, but not on either side more than the other. There was moderate tympanites but no dulness on percussion. He was somewhat under the influence of opiates. He continued in much the same condition for nearly a week, when the symptoms suddenly became worse, collapse soon appeared, and death in a few hours.

The *autopsy* revealed a fresh general peritonitis, a peritonitis of longer standing localized about the appendix, the intestines glued together by thick plastic deposits, a pocket of pus with fecal matter by the side of the appendix, and the latter perforated at its junction with the cæcum. The steps in the case were appendicitis, ulceration, perforation, extrusion of fecal particles, localized peritonitis and abscess, extension of the poison and the process to the general peritoneal cavity, general peritonitis, and death.

Many cases of supposed intestinal obstruction, such as intussusception and volvulus, with some evidence of inflammation, are instances of peritonitis from perforation of the appendix, or of abscess due to previous inflammation of the appendix and its results. The fallacious theory that often obtains is that an obstruction has occurred, which in a few hours has developed inflammation.

A case in my own hospital service well illustrates this error:

A youth of less than twenty years of age, walked into the hospital from a cab at the curbstone, on a summer morning, complaining of pain in the abdomen. He said he had been sick several days with this pain, which prevented his lying down—he must sit in bed, bent forward. His friends said it had been impossible at times to keep him in bed, his suffering was so great. The sickness had, it was declared, come on suddenly and he had repeatedly

vomited. After his arrival he vomited a yellowish fluid having a stercoraceous odor. He had considerable fever, a pulse of 130, a tense abdomen, and a look of desperate sickness. A consultation with my surgical colleague, Dr. Graham, resulted in the conclusion that the case was probably one of obstruction followed by peritonitis. Certain to die speedily without surgical relief, he was given the chance of laparotomy. Nothing but peritonitis was discovered. This was afterward found to have resulted from a perforation of the appendix.

It is probable that abscess rarely occurs until after perforation, which may be regarded as the direct cause of it. Inflammation of small surfaces of the peritoneum, however, often supervenes, and adhesions, as well as probably some degree of cellular inflammation, without perforation.

The mortality from appendicitis, considering the frequency of it, cannot be regarded as great. The mortality from those cases eventuating in perforation of the appendix, however, is great, since this is the condition that leads to induration and extra-peritoneal abscess; and to acute, severe, local peritonitis and intra-peritoneal abscess, or to general peritonitis. The general peritonitis is almost sure death, and the abscess, wherever it is, and however well it may appear to be surrounded by protecting plastic deposits, is a constant menace to life, as evidenced abundantly by its spontaneous opening into the abdominal cavity, the venous canals, the bladder and the chest cavity, as well as externally and into the intestinal canal.

In view of these considerations, it is of the highest moment that we should be able, if possible, to distinguish the mild cases from the grave ones. One-quarter at least of all post-mortems show recoveries from previously existing inflammation, or other disease of the appendix, and probably 15 per cent. show recoveries from peri-appendicitis. The duty and aim of the doctor must be to determine clinically, if possible, the cases that form this vast class and commit them strictly to medical treatment, while he insists on surgery for those that lead to death directly, or to the hazardous results of abscess. Can this determination be made with certainty?

The diagnosis of perityphlitis is usually easy. Localized pain, sometimes masked early by pain elsewhere—often in the epigastrium —and by vomiting; more especially some localized tenderness, soon some evidence both by palpation and percussion of tumefaction in the cæcal region, possibly pain in this quarter on flexing the thigh, with

some fever—all perhaps following or attending some diarrhœa—are ordinarily sufficient. Percussion dulness is often present over the tumor but not always, tympanites sometimes counteracts it completely, but palpation discovers an induration even of small size, if it is done carefully and in comparison with the opposite side. The diagnosis of acute appendicitis before the advent of perityphlitis is more difficult, from the absence of any tumor and the deep location in the abdomen of the tenderness. The appendix sometimes hangs down into the true pelvis and far from the anterior wall of the abdomen. An appendicitis may be ushered in by vomiting and pain in the epigastrium, which may evoke such complaint as to mask the pain in the cæcal region. In only a little over half of all cases of acute inflammation in the cæcal region is the pain mostly in that locality, while in nearly a third the pain is attributed to the abdomen generally.[1] Sometimes there is a misleading appearance of induration to touch due to the tension of the abdominal muscles over the tender point, a thing that disappears wholly under anæsthesia. For purposes of diagnosis the hypodermic needle is permissible for exploration, but only when it is absolutely certain that it may be passed into the centre of the induration without entering the general peritoneal cavity. It is unsafe to use it in a supposed intra-peritoneal abscess. The absence of fluctuation over an induration is no proof of the absence of pus; pus is present in all large and most small indurations of a few days' standing. In a few—it must be very few—cases digital exploration of the rectum will discover indurated tissue below the cæcal region.

A large proportion of first attacks, and probably many after attacks, of acute appendicitis will doubtless recover without abscess, and with slight cellulitis or local peritonitis and adhesions, under proper treatment, namely, quiescence in bed, hot applications, anodynes, light diet, and a rigid avoidance of all influences that can provoke general intestinal peristalsis. It can hardly be questioned that a majority of such cases even recover without these wholesomely safe measures of treatment and hygiene. Many cases of chronic appendicitis utterly fail to improve much under any treatment whatever, albeit treatment doubtless often prevents such from passing rapidly on to ulceration.

When to go beyond treatment of these diseases and insist on operation can probably not be determined by any absolute rule. Each case

[1] Fitz.

must be dealt with by itself to some degree, since cases differ widely, but there are certain general principles that ought to guide us, in view of which we cannot shirk our full responsibility.

Reliance on treatment is justifiable in acute inflammation in the cæcal region—*i. e.*, appendicitis, perityphlitis, or typhlitis—of moderate severity, in the absence of strong evidence of perforation, abscess, peritonitis, and of any considerable tender induration lasting two or three days without some sign of decrease, and high temperature, either continuous or recurring, rapid, weak pulse, or rapid, anxious respiration, surely attributable to this disease. But we can never know when a catastrophe is to occur, even in an apparently mild case such as is here characterized. A few cases falling in this category will suffer sudden perforation, general peritonitis, and death; but nearly all of them, failing of prompt recovery, will, if perforation occurs, have sharply localized peritonitis or perityphlitis, and probably abscess that will be easily discovered, and that will demand surgical treatment. That in a high percentage of a large number of post-mortem examinations made on all sorts of cases, there should have been found evidence of previous recovered appendicitis would seem to justify the position here taken.

Reliance on treatment is also justifiable in subacute and chronic inflammation where the constitutional symptoms are mild, pain and tenderness slight, and the induration small and not increasing. Such cases frequently suffer occasional slight exacerbations of short duration, which even do not positively indicate the need of an operation unless each recurrence increases the size of the tumor, or its sensitiveness to pressure, or develops other proof that it is an abscess. To say that every perceptible induration at the site of the cæcum should receive the ministrations of surgery is not justifiable; and probably numerous cases might be reported of operation on small indurations without discovering pus. One such is here presented.

Mr. A. B. entered my hospital service complaining of slight pain and tenderness in the right iliac region, and giving a history of several attacks of acute inflammation within a year. In each attack there was moderate fever, and frequently, at the beginning, diarrhœa. A small induration over the lower point of the cæcum was distinctly made out; it was slightly tender to pressure, and forward movement of the right thigh was embarrassed. This case was transferred to my surgical colleague, Dr. Parkes, in the belief that an operation ought to be made. He made laparotomy and found some thick-

ened omentum, numerous adhesions, some of which he separated—but neither abscess nor appendix. The wound healed promptly.

The two outlines given above include the vast majority of all cases, and if the premises are correct, then these cases are most properly committed to strictly medical treatment—and the only proper treatment is the conservative course already described.

But surgical interference is demanded in certain cases of inflammation in the region under consideration, whether they happen to be called typhlitis, appendicitis, perityphlitis, or by some other name, and the weight of first responsibility is on the physician more than the surgeon.

1. Surgery is imperative in cases of acute inflammation in the cæcal region with rather protracted high temperature and with distinct induration, sensitive to pressure, that does not show positive evidence of subsidence within two days, or three or four days from the beginning. This rule becomes more urgent if the induration continues to increase in size and sensitiveness after two days, or if symptoms of general peritonitis occur, or rapid, weak pulse, or rapid respiration. The vast majority of such cases left to themselves eventuate in abscess in less than a week, and many before that time lead to mortal peritonitis. Some require operation in less than two days from the beginning of the attack, and most of them have perforation of the appendix as early as the beginning of the symptoms.

I am aware that a few cases here characterized do not require operation, and would recover without it, but the number is so small compared to those in the greatest peril, to which it is a crime not to offer the benefit of surgery, and the danger of an antiseptic operation is so slight, that they constitute no impeachment of the rule.

2. Operation is required in cases of undoubted, severe, acute inflammation in the region of the appendix even though no particular induration is demonstrable, and in cases of acute localized peritonitis having its origin certainly at the appendix, and causing marked constitutional symptoms. In the one situation there is almost certainly such violent inflammation of the appendix as seriously to threaten perforation with all its dire possibilities; in the other perforation has occurred and an abscess is probably forming. In the one case there should be laparotomy and extirpation of the appendix; in the other laparotomy, extirpation of the appendix if possible, and treatment of

the abscess. Probably these two classes of cases cannot be distinguished from each other; many of the symptoms of the one belong to the other. Practically, it is hardly important that they should be distinguished, as both abundantly justify surgical interference; the patients are in vastly less jeopardy with the operation, when carefully made, than without it.

3. Surgery is specially promptly required in that small class of acute cases in which a large, sensitive induration develops rapidly, with high fever and general evidence of severe constitutional disturbance. Here extensive deposit and large abscess are almost certain to be present, and the danger of early rupture into the peritoneal cavity is considerable, hence the necessity of prompt action.

4. Surgical aid is demanded in all cases advanced to the subacute or chronic stage with distinct induration of considerable size, or with an induration that steadily increases in size for many days, since in most such cases pus is present. These are the cases where sometimes weeks and months after the acute stage a tumor of variable size is found in the iliac region, slightly tender, dull on percussion, and attended by a slight stiffness in walking dependent on pain in flexing the right thigh. Usually in such cases there are frequent exacerbations of moderate suffering in the affected region, often with slight fever, and disabling the patient for a day or two. Unless the tumor is very small, pus is almost invariably found in its midst, the quantity varying from a few drachms to many ounces. The patient cannot be safe so long as an abscess is present in the neighborhood of the cæcum, but the danger is in proportion to the size of the abscess. Some portion of the cæcum must generally form a part of the abscess wall, hence the liability of rupture into this canal and into the peritoneal cavity. In a case seen by the writer, in consultation with Dr. J. B. Murphy, a perityphlitic abscess had undoubtedly existed for a year, since the date of a previous sickness in which there were dull pain and fever for several weeks. The abscess ruptured into the abdominal cavity while the patient was scrubbing a floor; peritonitis rapidly supervened, a circumscribed large peritoneal abscess formed, which was opened by Dr. Murphy and a quart of pus evacuated. This case also exemplifies the large induration developing rapidly, referred to already. The apparently small difference between cases sure to die without operation and those likely to recover without it, as well as the

difficulty in finding the line of duty in management, is shown in the two cases that follow:

D. F. G., aged fourteen, robust and active, went hunting April 30, 1881, and got very tired. The whole of the next day he felt fatigued. May 2d he felt a little better, but had a mild diarrhœa; next day more diarrhœa, pain, and slight tenderness on pressure in the cæcal region. May 4th I was called and found the diarrhœa better, but still some pain and tenderness at the point mentioned; pulse was 100 and temp. 101° F. May 5th patient was improved; slight tumefaction in the cæcal region; pulse 100, temp. 101.7° F. May 6th, no pain; tenderness reduced; pulse 100, temp. 99.7° F. May 7th, temp. 99° F. There had been no increase in respiration rate and the patient felt convalescent. The next day at 11 P. M. a piercing pain was felt in the lower abdomen at the right side, lasting only a few minutes. Some tenderness was found soon afterward, but no tympanites; the pulse was 100 and small, temp. 98.5° F., respiration 20. Collapse came on in six hours; pulse was 150, temp. 98.5° F., and death in four hours more.

It was found post-mortem that perforation of the appendix had led to an intra-peritoneal abscess containing an ounce of pus; that the plastic wall of this abscess had given way at its superior point, a fulminating general peritonitis being instantly lighted up. The rupture was at the distal end of the appendix, which was gangrenous.

G. B., aged thirty, salesman, strong and active, had had occasional attacks of pain in the abdomen lasting for a few hours. He was seized last February, in the morning, with severe pain in the lower abdomen and went home and to bed. By evening his temperature was 104.5° F., then he discovered that the pain which had continued to some degree all through the day, was most in the right iliac region. His bowels were moved by an enema. I saw him first at this time, and found tumefaction and dulness in the cæcal region with the slightest possible tenderness; next day and the day after, the evidence of tumor was greater than at this time, the tenderness being reduced. The second day of attendance the temperature was over 103° F., the third day 102°, and the following day 99°, from which time convalescence was rapid and he was out in a week from the beginning. The pulse was at no time much above 100, and the respiration was not accelerated; there was no diarrhœa, and the patient was tranquil in mind.

It will be seen that the temperature rapidly fell after the third day of the sickness; with its fall the evidence of tumor rapidly disappeared. In this case tumefaction and dulness on percussion were marked in the cæcal region and entirely disappeared in the recovery, and there was never any notable tenderness. In the case of D. F. G. clinical evidence of tumor was slight, but during the first week there was con-

stantly more or less tenderness—a symptom we now know as some indication for surgical relief.

5. Surgery is justifiable in all cases of undoubted chronic appendicitis with occasional exacerbations, even if no induration is present. In all such cases we cannot doubt that the patient is in constant danger of perforation of the appendix, and mortal peritonitis or perityphlitis —a danger greater than that involved in an antiseptically done laparotomy and extirpation of the little organ entire.

If the diagnosis was positive in every case there could be no objection to this proposition, for it covers only cases of inflammation with some persistent cause, and not those with a temporary cause and a single seizure with no special tendency to a recurrence. The persistent cause is usually something within the appendix that makes pressure and irritation, and back of that some constriction of the appendicular opening, or some fault of its valve; the cases of one mild attack and quiescence afterward are those in which there is no incarceration of irritating matter of any sort, where the exit from the canal is free.

Fitz says catarrhal appendicitis is probably not recognizable. The diagnosis of such cases is beset with difficulties and probably many of them cannot be diagnosed, but some may be, and these should not fail to receive the benefit of surgery simply because they may not appear for the moment to be formidable. The diagnosis must often be made by exclusion, and is easier in males than females, since in the latter the pelvic organs are apt to produce symptoms resembling those of appendicitis. The following case is illustrative:

A lady of twenty-eight years, vigorous and robust, had experienced, from time to time since girlhood, slight pain in the right groin and thigh, often aggravated by exercise. Occasionally there was accompanying but less pain in the left groin and thigh. Last summer she had what appeared to be a dysenteric attack, passed bloody stools, had much pain in the abdomen, especially in the right side, and fever. She recovered and resumed her active life, which required her to be much upon her feet. Soon pain in the abdomen recurred, then came on slight fever, anorexia, occasional vomiting and constipation, tenderness over the hypogastrium and pain in the thighs. At the end of six weeks she entered my hospital service and was examined carefully, my gynecological colleague, Dr. Merriman, assisting. Then she was in bed, unable to move without pain in the abdomen, and suffering considerably in the thighs, hypogastrium, and right inguinal region. The abdomen was tender over its right side, from the border of the ribs to Poupart's ligament, but no tumefaction or dulness was discoverable. An examination of the

pelvic organs revealed nothing pathological, nor could any induration be felt through the vagina or rectum. She had a daily rise of temperature to 100° or 101° F. The thighs were kept flexed much of the time, pain in the psoas region occurred on flexing the right thigh against resistance; a daily enema was required; the appetite was poor and the sleep much disturbed by pain. She soon began to improve, and in a month or six weeks could turn in bed with only slight discomfort; her appetite was better, the pain less, and the temperature much of the time normal. The tenderness in the abdomen, however, persisted and became more marked in the cæcal region; and one or two tentative efforts at sitting up were followed by rise of temperature and return of pain lasting two days or more. It now seemed that the history and course of the case had excluded every other pathological condition but some chronic inflammation at the location of the cæcum and appendix, and that the time for surgical exploration had arrived, and my colleagues, Drs. Parkes and Merriman, examined her at my request and agreed that an operation was justified. Laparotomy was made by Dr. Parkes, and the appendix was found enlarged in its diameter, hard and tense, projecting forward in an erect position, and deeply congested. No other pathological condition was discovered in the abdomen. The appendix was extirpated and found to contain three small enteroliths and a quantity of thick, tenacious mucus; its walls were thickened. On laying it open longitudinally it assumed instantly a rolled form in the reverse direction—apparently the peritoneum contracted and the thickened mucous membrane and muscular tissue became extended. The patient made a good recovery from the operation and her symptoms have disappeared.

There was in this case no evidence of beginning ulceration of the appendix, and so no immediate danger of a perforation, but the tension of the organ was so great as to prove an almost complete closure of its opening, and must have led to ulceration sooner or later. Moreover, the patient was apparently a permanent invalid without the operation.

DISCUSSION.

Dr. I. E. Atkinson: In cases where perforation is clearly imminent, or has already occurred, all will admit that an operation is the proper procedure. Manifestly, however, the surgeon should be called in while there is yet hope of success from operative interference. On the other hand, it is certain that many cases of appendicitis and pericæcal inflammation recover without cutting, and that post-mortem examinations show, in an extremely large proportion of cases, evidences of old inflammations of these parts that have never been recognized during life, but which have recovered spontaneously. The fact that

such a large number of cases do not result in perforation, makes it most difficult to decide just when the operation is called for. Methods of diagnosis must greatly improve before a hard-and-fast rule for the management of these cases can be adopted. It would be highly injudicious for the physician to appeal to surgical interference upon every occasion when the inflammation may be definitely recognized. It appears to me that in view of the fact that a majority of cases of inflammation in this region recover, the physician, until he becomes more capable of recognizing the actual condition present, is compelled to assume the responsibility to postpone operation until it becomes reasonably certain that perforation is imminent. In order that the patient may not suffer from the delay, the physician should, in the given case, maintain an unceasing watchfulness, should keep his patient under constant close observation lest the favorable opportunity should pass.

Dr. R. H. Fitz: It is, of course, unnecessary for me to state that, in the main, my views agree with those of the reader. There are certain points, however, with regard to which I would say a few words.

In the first place, I should like to emphasize a conclusion already reached, that there must be many cases of appendicitis so mild as never to demand medical treatment. Although one-third of all post-mortem examinations may show lesions indicative of an appendicitis at some remote period in the patient's life, the clinical history will frequently give no evidence of any symptoms pointing toward a previous attack of this disease.

The paper on perforating inflammation of the vermiform appendix which I had the honor of reading before this Association at its first meeting, four years ago, was based upon the consideration of a large number of cases chiefly obtained from medical literature. The necessary criticism of such a consideration is that the cases are selected for publication and will not represent the experience of an individual. In other words, a personal experience, if sufficiently large, is likely to offer conclusions which may have a greater value than those derived from a much larger number of selected cases.

During the past four years it has been my fortune to see seventy-two cases of appendicitis. Most of these were visited in consultation; hence, in a certain sense, might be regarded as selected. Many of them were seen conjointly with my friends Drs. Homans and Richardson, whose surgical experience in this disease is already very large.

In the consideration of these cases the first point to which I will call attention relates to sex. In the paper already referred to, it was found that four-fifths of the patients were males. In my personal experience there are only twice as many males as females. The diagnostic value of sex is thus distinctly less than the collective experience claimed.

In this connection I may refer to the difficulties of diagnosis sometimes encountered. In general it is easy, and, above all, is based upon a localized tenderness and the exclusion of all causes for such tenderness excepting the appendix. Still it has been my misfortune to make a diagnosis of appendicitis when it did not exist, and to fail in recognizing its presence until after death

in a patient upon whom laparotomy was performed for the relief of a supposed acute, mechanical, intestinal obstruction. The failure to make a correct diagnosis was never serious, since it always led to the proper treatment of the existing disease. The following are the cases in which appendicitis was supposed to be present:

A patient of Dr. James B. Ayer, of Boston, was seen in consultation by Dr. J. Collins Warren and myself. A circumscribed peritonitis was recognized in the right lumbar and iliac regions, and, from its seat, sudden origin, and rapid progress, was thought to be due to an appendicitis. The cavity was successfully drained by Dr. Warren, but the patient died of acute pneumonia in the course of a fortnight. The peritonitis proved to have originated from an abscess of the liver, and this was attributed to a catarrhal colitis of long standing.

In a second case of erroneous diagnosis the patient, an athletic, young man, was seen with Dr. M. H. Richardson. There was a spreading peritonitis of the lower abdomen of sudden onset. The abdomen was opened and drained, but the appendix proved to be free from disease. After death the peritonitis was found to have resulted from a suppurating, subperitoneal, inguinal lymph-gland, the result of a gonorrhœa. Information concerning the latter disease was withheld till after the autopsy.

A third case was seen with Dr. A. T. Cabot. The history as obtained by us was so far misleading that a pyosalpinx was mistaken for an appendicitis. The tubes were removed by Dr. Cabot, and the patient was cured.

Finally, a girl of ten years, suffering from a sudden and spreading hypogastric peritonitis, with a deep-seated tumor below the navel, was seen with Dr. Homans. Although some doubt was entertained as to the appendicular source of the disturbance, a laparotomy was considered urgent. Dr. Homans successfully removed an ovarian cystoma, the pedicle of which had become recently twisted and its vessels strangulated.

Recurrence proved to be much more frequent than was found in the collective experience—44 per cent. in my cases against 11 per cent. in the larger series. It is interesting to note that the frequency of recurrence was about the same in those cases which were subsequently treated medically, as in those which were operated upon. So far as this evidence goes, a recurrent attack makes no more urgent demand for surgical treatment than does an original attack.

Recurrences might be many and severe, and the formation of abscesses during previous attacks was occasionally suggested by the severity of the symptoms and the length of the illness. Absolute evidence that an earlier attack may result in an abscess was furnished by a case seen with Dr. C. B. Porter. There had been previous attacks from which recovery by resolution had taken place. Dr. Porter operated and drained the peritoneal abscess, which was at the brim of the pelvis; the appendix was not isolated. After recovery recurrence again took place, and, finally, Dr. Porter removed the appendix after convalescence from a recurrent attack. It was thickened, neither ulcerated nor perforated, and was without a scar. It was adherent, to a limited extent, to the omentum. This case gives suggestive evidence that an

appendicular, suppurative peritonitis may occur without perforation of the appendix. It also shows that a subsequent attack of appendicitis may take place even when an abscess resulting from a previous seizure has been well drained and recovered from.

With regard to the duration of the disease, it may be said that although more cases recovered during the first three weeks under purely medical than under surgical treatment, this fact must be attributed to their mildness. Of the severe cases, quite as many demanded from four to eight weeks for complete recovery whether the treatment was medical or surgical. It cannot be claimed, therefore, for the surgical treatment of appendicitis that it has shortened the period of recovery. At least such a claim would not be justified by the results found in the cases which have come under my observation.

The rate of mortality in appendicitis is by no means clearly established. The physician who sees chiefly the mild cases, says that it is very low; while the surgeon who is called upon for aid in the gravest cases considers the mortality to be very high. So far as I am aware, the cases observed by me form the largest number reported in the experience of a single individual, and they may be considered as establishing, for the present, the rate of mortality. Of these cases 74 per cent. recovered and 26 per cent. died. About one-half of them were treated medically, the other half receiving surgical treatment.

Among those treated surgically 40 per cent. died, while of those under medical treatment 11 per cent. died.

The spontaneous evacuation of pus, "an event to be anticipated and guarded against," took place in 7 per cent. of the cases. This result is gratifying in comparison with the 28 per cent. of cases thus terminating in the collective experience previously recorded.

Of the cases treated medically 11 per cent. died, and in 14 per cent. there was the spontaneous evacuation of pus; thus in one-fourth of these cases the treatment should have been surgical.

Since these medical cases were about one-half of the whole number seen, it follows that the treatment should have been surgical in at least five-eighths of the cases.

The percentage of cases ending in resolution was 36 per cent., which is practically the same previously found. Medical treatment should therefore be limited to a little more than a third of the cases.

It is evidently desirable to make the rules for the surgical treatment of appendicitis as simple as possible, and I would advise it for

Urgent symptoms (rising pulse and temperature, increasing distention, and spreading pain) with or without a tumor; and for

A tumor, with or without urgent symptoms.

Recurrent attacks should be treated as first attacks. If recurrence is so frequent as to debar the patient from the enjoyment of life, or the ability to earn a living, removal of the appendix between the attacks should be advocated.

Dr. A. Jacobi: I desire to make only a few remarks, particularly in reference to the question when a case is to be "turned over to the surgeon." There

are some operations which ought to be done by every well-educated practitioner; such are tracheotomy, intubation, herniotomy, and the operation for perityphlitic abscess. The indications for the latter are not uniform, and the opinions of practitioners and writers differ widely. I am personally not inclined to think too little of rational medication and reasonable waiting, but in a case that occurred lately I was in favor of an immediate operation, the consulting surgeon was not, and was certainly correct.

I was called late one evening to see a man with perityphlitis. He had been under observation more than a day; was suffering from pain, and had a good deal of swelling. He had a pulse of 140, was weak and a little delirious. The people wanted to send for a surgical specialist to perform an operation. The surgeon said he would not perform the operation, but if the case were treated for six or eight days, and looked more promising, then he would. The doctor then applied ice day and night to the immediate neighborhood of the swelling, and treated the patient with opium. He got over his heart failure; the inflammation did not extend and in a fortnight afterward the same surgeon operated on the patient. If the operation had been performed the night I saw him first, that man would be dead.

As far as perityphlitis goes, I am positive it is very frequent. I am positive that the first attack does not always kill—it is the last attack. I have not seen a fatal case in which there were not positive signs indicating that the individual had a previous attack. I know this is contrary to the opinion frequently expressed lately. But why should it happen that the appendix should be the harbor of a foreign body as it so frequently is? There seems to be a reason; this is, that there is present a result of a former disease attacking the appendix, diminishing its elasticity, and facilitating the entrance of a foreign body (feces, or seed, etc.).

Five days ago a medical gentleman, from a neighboring county, called on me with a boy of eleven. The gentleman was as straight as an arrow, the boy was not. The father said the boy had what he believed to be an attack of peritonitis, the tenth or twelfth he had had in twenty months. It was a clear case of perityphlitis.

A boy, of fifteen, was taken with such a mild perityphlitis that I did not even think it necessary to puncture. He had another mild attack a few months afterward, and three months later, when I could not see him, he had a very serious attack with pain and high fever, and at one and the same hour he had perforation of the bladder and through the rectum. He did not get well without an extensive operation, in which part of the sacrum had to be removed to get at the abscess. If that boy had been punctured by me and operated on in time, he might not have had to undergo the severe operation afterward.

So I say there are quite a large number of cases in which we hardly know what to do. But I believe a puncture may be made in most cases.

Dr. Wm. Pepper: The statement of this matter by Dr. Bridge strikes me as altogether the fairest that I have heard or read. The drift of thought and opinion has been so strong of late in regard to operations, I am delighted to

hear these statements in favor of conservatism. I cannot protest too strongly against the recommendations of Dr. Jacobi that physicians should hold themselves responsible for operations in such cases. The operations I have been forced to do have been among the most terribly difficult operations I have witnessed, taxing the skill of the most trained laparotomist.

We have made great progress in recognizing these conditions and mapping out a line of treatment, and a great deal of that has been done by members of this Association. But we have not got to the point of establishing hard and fast rules.

Although, as Dr. Fitz shows, the mortality will be high under any plan of treatment, and although you are liable to errors in diagnosing, yet the medical treatment of cases of perforative appendicitis is so uniformly followed by fatal results that certainly the surgeon should be called in, and the responsibility at least divided with him. Our efforts must be directed ceaselessly to the detection and the formulation of early diagnostic points. It is not too much to hope that if a diagnosis can be established promptly, say within twenty-four hours, the results of early operations will be far more favorable than any yet achieved. In proportion as the diagnosis in any case is clearly established, does it seem better to take the risk of laparotomy.

DR. JOHN H. MUSSER: I rise, sir, to call attention to the relationship of local tuberculosis to cases of appendicitis and typhlitis, and in this connection to refer briefly to three cases which have come under my observation, which were of tubercular origin.

The first case that I shall report was one of perforative appendicitis with the formation of peri-appendicular abscess. The patient was a man, some twenty-two years of age, machinist by occupation. He stated, when I was asked to see him, that he had been particularly well prior to this attack, although his friends said that he had, in their eyes, lost some flesh. The attack, for which I was consulted, began acutely two days before with severe abdominal pain attributed to colic, with some nausea and with tenderness in the right iliac fossa. The pain was accompanied with diarrhœa. On the third day of my attendance a well-defined tumor was distinctly recognized in the usual locality, about an inch above the middle of Poupart's ligament, which could readily be recognized as due to pus. The formation of this mass was attended with a moderate degree of fever. My friend, Dr. Keen, saw him with me in consultation, and at once operated successfully. That there was communication with the bowel we well knew from the discharge of fecal matter. Without entering into detail I would say that the wound healed very promptly, and the patient recovered very well from the operation, and also from the local inflammation. His general condition did not improve, however, and, in spite of a most excellent appetite, he remained weak, and did not gain in flesh. Removal to the sea and all hygienic and dietetic advantages possible did not cause him to recuperate. A few weeks after the operation a most marked diarrhœa set in. Prior to attention having been called to this active diarrhœa the patient himself observed the occurrence of a couple of

loose stools each day. The diarrhœa could not be controlled. General tuberculosis, with all the phenomena seen, supervened, and the patient died of pulmonary and intestinal tuberculosis some four months after the operation.

I take it that the sequelæ of events in this case were, 1st, the occurrence of intestinal tuberculosis; one of the enlarged infected glands was situated at the mouth of the appendix, causing occlusion with retentive inflammation and the formation of an abscess. 2d, the tuberculosis, of course, progressed, but was not sufficiently far advanced to show marked symptoms during the convalescence from the operation.

The remaining cases were simply instances of perityphlitis tuberculous in origin. One of them, a woman, some thirty years of age, had a long course of remitting fever, at times intermitting in character, with local pain and tenderness, and with abdominal swelling. No tumor could be defined, but there was no doubt in the minds of Dr. Deaver, who had charge of the case, and of Professor Osler, who had seen the case, and during whose absence I had seen it, that it was one of local peritonitis. That it was tubercular in origin the sequence of events seems to prove, for more or less general infection of the peritoneum took place, and the pleura became involved with the occurrence of effusion, and finally involvement of the pulmonary structure.

The third case occurred in my own practice, and was also seen in consultation by Dr. Keen to advise regarding the probability of the necessity of performing a laparotomy. The patient, a lady of twenty-five years, presented the usual symptoms of perityphlitis. No well-defined tumor could be ascertained, but the general local symptoms warranted the suspicion of a local abscess. Operation was not decided upon, however, and the most protracted convalescence, attended with emaciation and attacks of regular fever, was interrupted by the development of diarrhœa, peritonitis of subacute character, followed quickly by pulmonary tuberculosis, with death on account of the involvement of the lung structures. The autopsy showed the above to be about the course of the pathological events.

I am quite in accord with those who are conservative in regard to the treatment of recurring typhlitis or appendicitis. I have had cases under my care which I attended in a number of attacks, and I am quite satisfied, particularly as each one is lessening in severity, that I have never had any of them operated upon. Some of them have extended over a period of many years.

VARICOSE ANEURISMS OF THE AORTA AND SUPERIOR VENA CAVA.

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THERE is, perhaps, no pathological condition whose symptoms are usually more characteristic than are those of obstruction to the normal flow of blood in the superior vena cava. Such a condition may be brought about by compression by tumors or aneurisms, by inflammation and consequent thrombosis of the vein itself, by rupture of an aneurism of the aorta into the vena cava, by penetration of a malignant growth into the vessel, or by bands of inflammatory tissue constricting it from without.

The characteristic symptoms consist in intense œdema and cyanosis coming on rapidly or slowly, and limited to the upper half of the body; together with such secondary symptoms as would naturally follow these conditions. It is difficult to conceive of any cause suddenly operating to bring about obstruction, yet failing to produce these effects. Only when the cause operates very slowly does it seem possible that the symptoms may be slight, or even absent, the collateral circulation having become established.

Rupture of an aneurism of the arch of the aorta into the superior vena cava, one of the causes referred to, is so rare an occurrence that we have been interested in looking into the history of the affection, in connection with a case occurring under our own observation, with the hope of determining whether there exist any symptoms which would permit of a diagnosis during life.

The following history of the case is imperfect in some points, owing to the patient's slight knowledge of English, as well as to the difficulty which he experienced in talking, on account of the great dyspnœa from which he suffered:

Tak Sing, Chinaman, aged forty-eight years. Was admitted to the Hospital of the University of Pennsylvania, September 22, 1888. His parents are both dead, of causes unknown. Nothing else can be elicited in the way of family history. He has been a cook on a man-of-war, and since 1876 has been working in a laundry. Although on close questioning he says he has noticed numerous small varicose veins over his thorax for four months, he claims that he has always been a healthy man until four weeks ago. At this time he had been working very hard in his laundry, and sleeping in a cold, damp cellar for several nights, after which he was attacked by a severe pain in the chest, which prevented him from sleeping. He was also unable to lie down at night, presumably from dyspnœa. He noticed that his neck and shoulders were swelling, the left side being more swollen than the right. After this the chest became swollen, and he says that his feet were so likewise. The notes of the case make no reference to the condition of the arms. It could not be positively determined whether œdema first appeared at this time, or whether some had existed four months before, at the time varicose veins of the chest were first noticed.

A week before admission he began to have difficulty and pain in swallowing, and could only take milk and soup, and occasionally a fragment of meat. A Chinese physician was summoned from New York, and applied the moxa to both sides of the neck, the shoulders, and the elbows, but his condition continued to grow worse. Three days ago a watery discharge from the left ear began. He has had no trouble in emptying his bladder, though he has passed less urine than usually. He has had some cough with expectoration since he was first taken sick.

When first examined by us he was found suffering from extreme dyspnœa and complaining greatly of a burning sensation about the chest; but whether this was felt internally or on the surface of the skin could not be discovered. He sat erect or leaning forward in bed, entirely unable to lie down, and able to sleep but little. The eyes were suffused, the temporal veins congested, the tissues of the face cyanosed and very œdematous, the tongue clean. The neck was symmetrically and very greatly swollen, pitting and tender on pressure. No enlarged glands could be detected in it. The tissues of the chest were everywhere œdematous, giving the impression that the patient was a stout man. The surface of the thorax was covered with numerous small varicose veins. The arms, forearms, and hands were œdematous, and somewhat cyanosed. The pulse was small, well filled, and the tension possibly slightly increased.

The œdema ceased entirely below the thorax, without any sharp line of

demarcation, and the hips and legs were those of a slightly built man. The feet were cold.

The astounding contrast between the size and appearance of the upper and lower parts of the body would have been ludicrous had not the extreme distress of the patient removed any feeling but that of pity.

The supra-condyloid and inguinal glands were distinctly enlarged.

The examination of the lungs was unsatisfactory on account of the extreme œdema of the subcutaneous tissues. The respiration was costo-abdominal and symmetrical. Anteriorly the right side seemed everywhere duller than the left, but there was no impairment behind. Vocal resonance was normal. Numerous fine mucous râles could be heard during inspiration over both bases posteriorly, though more on the right side, and with slightly prolonged and bronchial expiration. In front on the right side there were a few fine râles, heard chiefly on inspiration, and the respiratory murmur was feeble. Auscultation of the left lung showed nothing amiss. The determination of the cardiac dulness was also unsatisfactory on account of the œdema, but it appeared to extend from the third rib downward, and laterally from the right border of the sternum to the nipple line. The apex-beat was in the normal position, and was rather forcible. A thrill could be felt over the apex and base. Auscultation at the aortic cartilage revealed a loud, musical murmur of a very peculiar character, reminding one of the sighing of the wind. Being loudest and highest pitched with the cardiac systole, it died away very considerably during the diastole, and lowered its pitch by several tones, to rise again both in volume and pitch with the next systole. It was thus *continuous*, and had a distinctly venous quality, although unlike a venous hum in that it was distinctly rhythmic. The systolic portion of this murmur was transmitted very distinctly under the right clavicle, and, indeed, everywhere over the right chest, both in front and behind, and also into the vessels of the neck and down the arm as far as the elbow. It could be heard faintly and distinctly at the pulmonary and xiphoid cartilages, but not at the apex. The abdominal organs appeared to be normal.

Dr. C. M. Hay, at that time Resident Physician in the Hospital, made careful notes at short intervals during the time the patient was under observation, and abstracts from some of these show the course of the disease:

September 25. The patient's condition is practically unchanged. Nitroglycerin has been given several times without effect, but he now appears to feel better after purgation with elaterium.

26*th*. Urine is acid, highly colored, quantity small, albumin in moderate amount, specific gravity 1022. No casts, blood, epithelium, or pus.

28*th*. In spite of the elaterium and pilocarpine the œdema and cyanosis are more extreme. The patient sleeps very little, and breathes with considerable difficulty, and has attacks of coughing. The pulse is weak and irregular; that

at the right wrist appears stronger than, though synchronous with, that at the left. The œdema, however, interferes with an accurate examination. The murmur is the same as at first. Respiration on the right side is fainter than on the left, and the percussion on this side seems everywhere duller.

30*th*. General condition the same. He complains of great burning in the throat and chest, especially on swallowing his whiskey.

October 1. Expectorated this morning about a tablespoonful of blood-stained mucus. Swallows considerable food, but with pain and difficulty. The chief complaint still is of the burning pain in the chest, aggravated by swallowing whiskey. There is a faint systolic murmur heard at the apex, and probably transmitted to this spot. The venous murmur at the aortic cartilage and under the clavicle is plainer, and the loudest portion of it almost synchronous with the first sound of the heart.

3*d*. The œdema is greater. The left arm is more cyanosed and œdematous than the right, being a ½ inch greater in circumference at the elbow.

5*th*. Dropsy and cyanosis of the upper half of the body still greater, the swelling being more marked on the left side of the face and neck and in the left arm than on the right side. The left forearm measures 1 inch more than the right. No œdema of the lower part of the body. The patient still complains of intense burning pain after each dose of stimulant. He is subject to attacks of stupor of short duration, and at times is very irritable.

6*th*. The œdema is still greater in the left arm, but the right arm is much less swollen than it was a week ago. The œdema of the chest is slightly better. The right side of the chest anteriorly is still duller on percussion than the left. Posteriorly there is no material difference. No evidence of pleural effusion. Respiratory sounds normal on both sides. The venous murmur is of the same character as before, but is of a little lower pitch, and is loudest at the end of inspiration. The patient has for a week been taking iodide of potash, tincture of nux vomica, and tincture of digitalis; also codeine and pilocarpine p. r. n., and dry cups over the chest at intervals.

9*th*. There is to-day some œdema of the scrotum.

10*th*. Condition worse. Cyanosis very extreme. Swelling of the scrotum has disappeared.

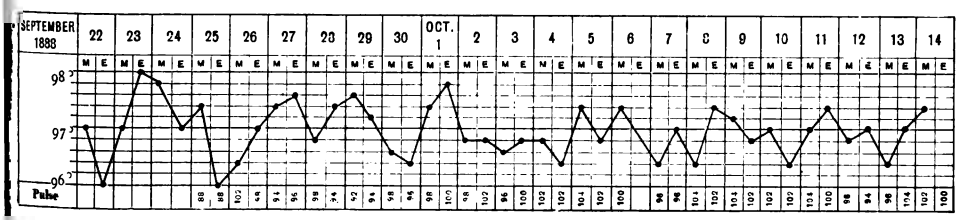
12*th*. Dyspnœa more marked and respiration labored and noisy, expiration being especially prolonged and stridulous. There is undoubtedly an area of percussion dulness on the right side, extending downward to the upper border of the third rib, and transversely toward the left as far as the left edge of the sternum. The respiration on the right side is much louder and fuller than on the other, but in other respects there is nothing worthy of note. The heart sounds cannot be distinctly heard on account of the noisy respiration, and the venous murmur is not heard either at the aortic cartilage or under the right clavicle. Examination of the throat made by Dr. Reeves shows the half arches of the palate covered with a muco-purulent secretion, and the larynx filled with the same. There seems to be a partial paralysis of the right abductor muscles, but the difficulty attending the examination renders the observation

uncertain. There is diffuse redness and extreme congestion of the whole of the interior of the larynx, with a diffuse œdema of the larynx and trachea. The patient being in great agony, dry cups were to-day applied with marked relief. The presence of occasional stupor and of irritability is still noticed, and in a few instances he has seemed to be wandering in his mind, though it was not possible to determine this with certainty.

13*th*. The expectoration is distinctly more frothy and serous, and less purulent than during the preceding week, and contains to-day slight traces of blood. The patient breathes with much difficulty.

14*th*. The œdema has increased greatly in the entire upper half of the body. Respiration is stridulous and very labored, but has been relieved slightly by dry cupping. Numerous large and small mucous râles are audible throughout the chest, especially on the right side.

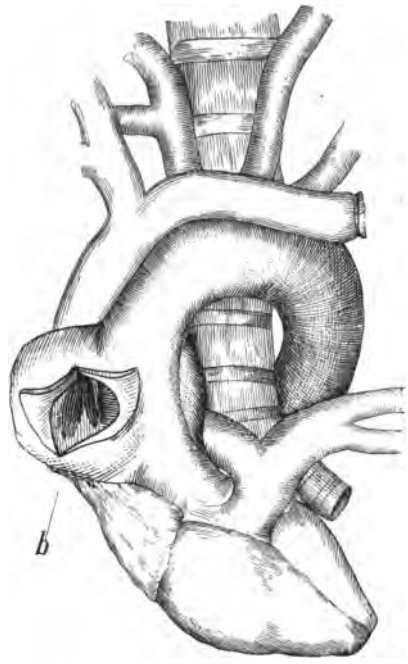
15*th*. At about noon, yesterday, blood oozed from the conjunctiva, and the sputum was tinged with blood. At 5.30 P.M. he had an attack of choking, and expectorated with great difficulty about f℥ij of bright, arterial blood. At 6 P.M. he became very drowsy, and gradually grew more stuporous and unconscious. The pulse began to fail, the cyanosis became almost black. Cheyne-Stokes respiration developed and continued until death at 8.45 P.M.



The temperature throughout the disease tended to be subnormal, as shown in the accompanying chart. Only once did it reach an elevation of 98° Fah.

Autopsy three hours after death.—Man below medium height. No rigidity or ecchymoses. Cyanosis much less marked than during life. Upper half of the body œdematous. No œdema below the waist. Lower extremities emaciated and shrivelled. On incision through the skin there is profuse venous oozing. On opening the thorax the tissues of the anterior mediastinum appear matted together, and everywhere œdematous. Pericardium contains about an ounce of bloody serum. Heart as a whole not hypertrophied. Valves normal. Right auricle large, and its walls look thicker than normal. The index finger enters the superior cava from the auricle for slightly over 1 inch, but then is tightly compressed. The inferior cava admits two fingers to the first joint. The coronary arteries are pervious and not calcified. The aorta is a little dilated, and 1½ inches from the aortic valves is an orifice the size of a half-dollar on the postero-lateral aspect of the convexity of the arch. This opening looks into an aneurismal sac projecting to the right and posteriorly and of the size of an orange (see figure). The tissues around the sac are

thickened. It is bounded on the right by the right lung, which it compresses, and to which it is adherent. Posteriorly it lies against the trachea and œsophagus and the right bronchus. The sac has everywhere thin walls, and is full of dark, moderately firm, non-laminated coagula.



The subclavian and innominate veins are pervious. At the junction of the right internal jugular and right subclavian veins is a globular thrombus the size of a large pea lying behind one of the valves. The upper part of the superior cava is much thickened, and is compressed by the aneurismal sac, which lies directly upon it. At its termination in the venæ innominatæ it does not admit the tip of the little finger passed into it from above, and this narrowing continues throughout the course of the vein to a distance a little over 1 inch above the auricle. A probe passed through the vein from above enters sometimes into the auricle, sometimes directly into the aneurismal sac. The œsophagus is tightly compressed by the aneurism, and the trachea is flattened in a lateral direction from the right side. The aneurism laid open shows within it and passing through it the thickened and compressed superior vena cava, and in the wall of the latter an irregular erosion ¾ inch in length in a vertical direction, beginning about 2 inches above the aneurism, and involving one-half of the circumference of the vein. The edges of this opening are irregular but smooth, and unoccupied by coagula. A probe passed from the

right auricle enters the aneurismal sac through the same opening. Just above the bifurcation of the trachea, at the point where this tube is flattened, there is an erosion of the cartilage, and possibly a small, though not discoverable, communication with the aneurism, since much blood is present in the trachea and bronchi. The recurrent laryngeal nerves are uninvolved. The lungs show extensive fibroid adhesions to the costal pleuræ on both sides, especially at the bases. There is no pleural effusion. The bases of the lungs are collapsed. At the apex of the right lung there is a cavity the size of a marble. The kidneys are distinctly fibroid, and the capsule slightly adherent and thickened. The brain reveals nothing of importance. The azygos vein does not appear to be dilated. The opening of this vein into the vena cava lies above the commencement of the constriction, and cannot be discovered.

An extended search through medical literature has brought to light only 28 cases of this lesion, including one (Case XVIII.) in which the symptoms were quite typical, but in which recovery took place. With our own case the total number, therefore, equals 29. Brief abstracts of the salient features in the clinical histories of these cases are as follows:

CASE I.—Reported by Beevor. *Lancet*, 1832–33, i. 800, and ii. 63.

Previous history: Not given.

Symptoms following the rupture: Enormous turgidity and varicose condition of the veins over the chest, which inosculated freely with the epigastric veins, etc. Œdema of the head and upper extremities [and chest?]. Auscultation revealed the presence of an aneurism. Man very rapidly sank, and latterly had terrified notions about his situation, thinking he was among thieves, men of blood, murderers, etc.

Duration of symptoms following rupture: Not stated.

Autopsy: An aneurism of aorta, capable of holding a pint of fluid; had produced interstitial absorption of sternum. Vena cava quite impervious for ½ inch, beginning 1 inch above right auricle; and just above the obliteration was a rent in the coats, which led directly into the enlarged aorta beneath. The azygos greatly dilated; entered cava just above the point of obstruction. At entrance of left subclavian [innominate?] was another and larger rent leading also into the aorta, and constituting a second communication between arterial and venous system. Heart normal.

This case is quoted by Peacock (*Trans. Path. Soc.*, Lond., xix. 127) as identical with the following. They are, however, distinct, as the first, with autopsy, was reported March 5, 1833, while the second patient did not die until August 4, 1833.

CASE II.—Reported by Thurman. *Med. Chir. Trans.*, 1840, xxiii. 323. See also *Lancet*, 1832–33, ii. 666.

Previous history: Coachman, aged forty-one; intemperate; much exposed to wet and cold. For a long time cough, dyspnœa, and palpitation.

Symptoms following the rupture: For a few days pain in neck and shoulders, replaced by swelling and purple color of face, then, in a few days, of chest, especially on right side, and of right arm. Clusters of veins, almost varicose, over chest and back. Pulse 100, rather hard. Sense of weight and stiffness over shoulders. Dizziness, stupor, confusion of mind. Dulness on percussion, distinct impulse, and vibratory murmur under right clavicle and to right of upper part of sternum. Same murmur, not so loud, over right carotid and origin of aorta. Some crepitation over both sides chest behind, especially above. Œdema became extreme; spread to left arm, and slightly to ankles and scrotum some days before death. Cough worse; dyspnœa finally extreme; expectoration difficult and tinged with blood. Toward end delirium almost constant.

Duration of symptoms following rupture: About 2 months.

Autopsy: Heart large; ventricles dilated; valves normal. A diffuse, true aneurism of ascending aorta, size of fist, commencing with pericardium and extending for 1½ inches above the valves, principally on right side. Vena cava behind aneurism; a round perforation ¼ inch across and with smooth edges, ½ inch above entrance of azygos vein, opened into sac. Lungs crepitant. Pleural adhesions both sides.

CASE III.—Reported by Reid. *Edinb. Med. and Surg. Journ.*, 1840, liii. 95. Specimen also studied by Thurman. *Med. Chir. Trans.*, 1840, xxiii.

Previous history: Tinsmith, aged thirty-five; intemperate; long been subject to palpitation.

Symptoms following the rupture: After lifting heavy weight had uneasy sensation in chest as though something giving way. Soon œdema and lividity of upper part of body, and confusion of mind. When examined after three weeks showed great œdema and lividity of arms and chest, and especially of face and neck. Conjunctivæ injected. Much dyspnœa; some cough and difficulty of deglutition. Urine scanty. Heart's impulse strong; apex depressed. Double bellows murmur over cardiac and sternal regions, especially upper part of latter; the systolic portion more prolonged, the diastolic shorter and sharper. Extended dulness over heart and upper part of sternum. Later œdema increased, with slight swelling of scrotum. Orthopnœa; delirium; died suddenly.

Duration of symptoms following rupture: 4 weeks.

Autopsy: Aorta at origin suddenly dilated, forming aneurism size of fist, limited to sinuses of Valsalva. Projected into anterior and left part of right auricle, and here it had termination of superior vena cava stretched over its outer surface. This portion of the auricle and the termination of cava communicated with sac by two oval openings with defined, rounded edges. Would barely receive tip of little finger. Cava pervious, but must have been compressed by the aneurism. Heart hypertrophied; left ventricle dilated; valves healthy; pericardium universally adherent. About a quart of serum

in each pleural cavity. Lungs compressed, small; contained considerable blood and frothy serum.

Though this is really an instance of perforation into the right auricle, the superior cava was involved as well, and the case may, therefore, with propriety be included here, particularly as the symptoms were purely those of rupture into the cava.

CASE IV.—Reported by Young. *Edinb. Med. and Surg. Journ.*, 1841, lv. 63.

Previous history: Gentleman, aged fifty-six; temperate; well until attack of rheumatism. In a year another attack, and at same time loud ringing cough lasting some weeks.

Symptoms following the rupture: Some months later, without premonitory symptoms, suddenly taken at night with deep blackish-purple color and great swelling of face, neck, and chest as far as second rib. Veins of forehead and temples very prominent. Headache. No dyspnœa. Next day respiration rather difficult; great restlessness; headache persisted. Pulse small and compressible. Over middle of sternum an impulse stronger than that over heart. No thrill or murmur anywhere. Chest had become œdematous, which hindered percussion being employed. Dyspnœa became excessive, and great dysphagia developed; both apparently due to swelling in neck. Respiration bronchial in back, with râles. No further examination of heart, probably on account of excessive restlessness. Patient talked incoherently. Death.

Duration of symptoms following rupture: About 2¾ days.

Autopsy: Livid color almost entirely gone, and œdema much less. Pericardium adherent to sternum. Heart not hypertrophied; valves normal except aortic, which was thickened, and orifice dilated. Aneurism of ascending portion and of arch, holding 1¼ pounds. Vena cava stretched over sac and adherent to it; a perforation in vein over ½ inch in length, with retracted, thin and irregular edges, and situated at about the level of the entrance of azygos vein; opening into right wall of aneurismal sac.

CASE V.—Reported by Law. *Dublin Med. Journ.*, 1842, xxi. 433.

Previous history: Man, aged forty-two; always well up to onset of the symptoms.

Symptoms following the rupture: Took a morning walk, and, not feeling well, lay down; face suddenly became swollen and dark purple. When seen next day, face, upper part body, hands and arms deeply livid; face, neck, upper part chest very much swollen. Suffusion and serous infiltration of eyes. Veins of neck hard and cord-like. Heart-sounds heard beyond ordinary limits, especially toward right subclavicular region. In other respects heart normal. Examination of lungs negative. Repeated examinations of heart and lungs gave no other results. No pain; but distress, discomfort, oppression. Very feeble pulse at wrists; strong in femorals. Subjective symptoms increased. Intellect became torpid, but no coma, and patient died suffering.

Duration of symptoms following rupture: 6 days.

Autopsy: Aneurism size of small orange from posterior and right side of ascending aorta. Beginning 1 inch above its entrance into heart, the walls of the vena cava were thickened for about 1 inch, and closely approximated by the pressure of aneurism. At this point was an unobstructed circular perforation about 2 lines in diameter opening into sac. Partial adhesion between opposite sides of vein. Aortic valves normal.

CASE VI.—Reported by Cossy. *Archives Gén. de Méd.*, 1845, ser. 4. t. ix. 33.

Previous history: Woman, aged forty-five; began fourteen years before to have slight attacks of palpitation and oppression. These gradually increased in intensity and duration. Finally palpitations became painful, but did not interfere with her work.

Symptoms following the rupture: Without further premonitory symptoms woke one morning with dizziness and some swelling, and violet color of face and right arm. Two hours later extreme dizziness and consciousness lost for several minutes. By evening face and arms greatly swollen, the right more than the left. Swelling increased. Examination ten days later showed face, arms, and neck much swollen and intensely violet; right arm larger than left. Chest not affected. Giddiness; sense of painful tension in head; continual noise in right temple and ear. Numerous veins outlined on face; none seen on arms. A distinct shaking of chest with heart's impulse, but without heaving or thrill. Heart seemed enlarged. A very loud, long, widely diffused, rasping, systolic murmur heard with maximum intensity under sternal end of right clavicle. Here complete dulness on percussion, but no pulsation or thrill. Posteriorly murmur loudest in right infra-spinous fossa. Above internal half right clavicle a pronounced systolic pulsation and thrill extending into vessels (veins) of neck of this side only. Radial pulse feeble, narrow, synchronous on two sides. Dry cough; slight dyspnœa; no râles. Dulness and feeble breathing behind on right side below. Later veins of forehead and the jugulars became a little more prominent. Sleepiness and then coma developed.

Duration of symptoms following rupture: 11 days.

Autopsy: Brain very pale. Hydropericardium. Hydrothorax both sides. Heart hypertrophied; valves normal. An aneurism size of hen's egg situated on convex portion of ascending aorta below origin of innominate. Vena cava on its posterior wall and adherent to it. A perforation 9 mm. high and 5 mm. broad, situated 15 mm. below entrance of innominate vein, opened into aneurismal sac. Cava only 26 mm. wide here, but below, at entrance of azygos, was 55 mm. wide. Azygos not dilated.

CASE VII.—Reported by Mayne. *Dublin Quart. Journ. Med. Sci.*, November 1, 1853, 257.

Previous history: Woman, aged fifty; laborious work. Well until forty-fourth year. Then dyspnœa, cough, and palpitation on going up stairs began. Had had since then several attacks of severe oppression, relieved by venesection. Symptoms grew worse, prevented regular work. Stooping

especially bad, as it produced dyspnœa and swelling of eyelids, face, and hands.

Symptoms following the rupture: While stooping suddenly felt as if strangled; persistent, intense feeling of suffocation; giddiness; remarkable change in color of face; breathing greatly embarrassed; absolute orthopnœa. Examination next day showed deep plum color and great œdema of face, neck, shoulders, and upper part thorax. Veins enormously distended, and in many places varicose. Eyes seemed starting from head, and showed extensive sub-conjunctival œdema. Occasional short cough without expectoration. Pulse jerking, like aortic regurgitation. Dulness on percussion bounded by left edge of sternum and line from sternal third of right clavicle to within 1 inch of nipple. A very strong heaving systolic impulse in second and third right intercostal spaces; vastly greater than at apex. Over impulse a very distinct thrill, and a remarkably loud, single, systolic, widely-diffused superficial bruit; maximum at second right costal cartilage. So loud that heart's action could not be accurately explored. Respiratory murmur inaudible over upper part right chest in front. No râles anywhere. Purring tremor felt in right subclavian and internal jugular veins. None in left. Temporary improvement followed venesection; then obstinate vomiting, stupor, drowsiness, delirium, convulsions, death.

Duration of symptoms following rupture: 8 days.

Autopsy: Congestion and œdema of parts supplied by superior cava. Right internal jugular nearly as large as small intestine. Serum in brain cavities. Lungs healthy; their mucous membrane very vascular. Heart healthy. Aneurism of aorta in shape of enormous dilatation occupying entire arch and about 2 inches of descending portion. Right and left innominate veins and superior cava adherent to surface of sac. Left innominate and cava considerably constricted where adherent; but below, from point of entrance of azygos, cava of normal size. Opening size and shape of shirt button-hole on right side of sac into vena cava; its edges sharp and irregular, as if recently made.

CASE VIII.—Reported by Goupil. *Thèse de Paris*, 1855, No. 50, vol. vi.

Previous history: Cavalry officer, aged fifty-eight; always some shortness of breath, which had increased during last twelve years. Odema of lower extremities at times for several years.

Symptoms following the rupture: After fit of anger lost consciousness. This followed by violent palpitation and excessive dyspnœa, with rapidly increasing œdema, cyanosis, and dilatation of veins of upper part of body. At base and at right side sternum a soft, prolonged, systolic murmur, and a stronger and harsher diastolic murmur. Suffocative attacks, restlessness, headache, and attacks of fainting developed. Pulse rapid, small; heart's action violent; death.

Duration of symptoms following rupture: 1 month.

Autopsy: Left ventricle hypertrophied. No disease of valves. Aorta atheromatous; became dilated immediately above the valves. An aneurism, size of hen's egg, began 5 cm. above the valves, and, projecting backward

and to right, pressed on right auricle, superior cava, right bronchus, and pulmonary artery. Vena cava compressed; and just below entrance of azygos vein an oval perforation into aneurism, 35 mm. in circumference, with irregular, red, eroded edges. The portion of vena cava below the entrance of azygos flattened and much smaller than above. Azygos vein dilated. Double hydrothorax. Lungs compressed.

CASE IX.—Reported by Tripier. *Thèse de Paris*, 1863, p. 63. Quoted by Peacock. *Trans. Path. Soc.*, London, 1868, xix. 140.

Previous history: Travelling merchant, aged fifty-five; temperate; always well except occasional dizziness.

Symptoms following the rupture: Taken suddenly at night with vertigo and dyspnœa, followed by very marked swelling and lividity of head, neck, face, arms, and upper part trunk, with distention of veins. Nothing remarkable about respiration. Sounds of heart normal; but 3 cm. from right side sternum beneath clavicle was dulness, pulsation; soft, feeble systolic murmur. Pulse small and frequent. Great agitation, followed by delirium and lapsing into coma, with increased evidences of obstruction of venous circulation. Death.

Duration of symptoms following rupture: 15 days.

Autopsy: Aneurism, large as fist, egg-shaped, projected to right and posteriorly from aorta above the valves. Adherent to pulmonary artery and to vena cava. Vena cava, though flattened and contracted where in contact with aneurism, was permeable throughout, and again expanded below. Was a perforation into aneurism of button-hole shape, 2 cm. long, situated 2½ cm. from commencement of vein. Its edges slightly broken, and very fine and close together. Fluid in pericardium. Slight hypertrophy of heart. No disease of valves. Great congestion of membranes and substance of brain.

CASE X.—Reported by Eastes. *Med. Times and Gaz.*, 1864, i. 393. See also report by Gull. *Lancet*, 1864, i. 409.

Previous history: Arsenal laborer, aged thirty-four; cough and dyspnœa more or less since Crimean war, and carrying load always caused cough and soreness across chest.

Symptoms following the rupture: About five weeks before seen, face, neck, and right arm began to swell and grow purple; left arm slightly so. Dyspnœa much worse, especially on exertion. Examination showed great cyanosis and œdema of upper half of body; veins of thorax engorged; cough severe, with frothy sputum. Decided dyspnœa; no dysphagia. Pulse small, regular, weaker on right side. Diastolic thrill over right side chest. A soft, whizzing, and very distinct murmur of venous character over aorta; diastolic at commencement of arch, but both diastolic and systolic, but chiefly the former, passing upward and over middle of arch. Maximum intensity at third right costal cartilage. The murmur had a continuous churning sound, like that in enlarged vessels in thyroid gland. No sound in right side neck. Percussion normal. Coarse crepitation at bases, both lungs posteriorly; bronchial breathing at right side in front. Dyspnœa, cough, and œdema increased. Erysipelatous inflammation set in. Death.

Duration of symptoms following rupture: About 7 weeks.

Autopsy: Aneurism, size of fist, on right and posterior wall of ascending aorta; its mouth extending from 1 inch above valves to innominate. Superior cava lay in front of sac. At its back part, just above auricle, was a perforation into sac; size of lead pencil and with smooth edges everted toward the vein and evidently not recent. Aorta atheromatous. Heart of natural size. Lungs crepitant; much mucus in the tubes. Fresh pleuritic adhesions between right lung and pericardium. Recent pericarditis with effusion.

CASE XI.—Reported by Gallard. *L'Union Méd.*, 1865, xxvii. 564.

Previous history: Basket-maker, aged sixty; always well except for attacks of oppression with cough at times, terminated by abundant expectoration.

Symptoms following the rupture: Suddenly in middle of night extreme difficulty in breathing and a violet coloration of face. When seen next day very marked cyanosis of face, neck, arms, and upper part of trunk, with some coldness and œdema. Cyanosis became intense. Injection and some chemosis of eyes. Subcutaneous veins swollen without pulsation. Œdema of entire upper half of body developed, alike on both sides. Dulness extending under sternum to under right clavicle, and a vibratory thrill over dulness. A widely-diffused systolic and diastolic murmur; its maximum intensity under right clavicle at the position of the dulness, where it was strong and rasping. Weak at apex; heard under clavicles and in carotids; heard down back. Its maximum intensity in back was at right of fourth dorsal vertebra. Pulse same force in two radials. Voice a little rough; respiration frequent, labored, a little noisy. No dysphagia. Next day unconscious; stertorous; very great œdema; general coldness; insensibility of skin and mucous membranes; feeble, rapid pulse; death.

Duration of symptoms following rupture: About 2 days.

Autopsy: Ascending and transverse portion of arch very much dilated. The ascending portion had sacculated aneurism. Vena cava compressed and adherent between aneurism and arch of aorta; so narrow that a uterine sound could hardly pass. Perforation into sac, size of a silver 20 centime piece, situated just below entrance of azygos vein. A clot contained in aneurism obstructed it and projected through it. Trunk of azygos slightly flattened, compressed by calcareous gland. Innominate and azygos veins voluminous and engorged. Aorta atheromatous. Nothing to note in heart.

CASE XII.—Reported by Hayden. *Dublin Quart. Journ. Med. Sci.*, 1866, xli. 434.

Previous history: Mason, aged thirty-three; intemperate; always well; strained himself in lifting heavy stone, and felt acute pain in dorsal region and could not work for some days. Two weeks later felt sudden faintness and a weakness in limbs; passed off in few minutes. About this time seen at hospital, and slight hoarseness and cough noticed.

Symptoms following the rupture: In three weeks more seen again, and presented the symptoms of the lesion. Deep cyanosis and œdema of entire

upper part of body down to diaphragm. Conjunctivæ injected with dark venous blood. Especially face and neck deep purple. No thrill in veins of neck. Chest covered with tortuous veins in groups. Temperature of upper half body low, and man complained of chilliness in that part. Pulse small, regular, rapid. Cough and expectoration. Respiration 24. No dysphagia. Percussion less resonant to right of sternum from clavicle to nipple, and respiration feeble here and down to infra-spinous fossa behind. Elsewhere loud, with muco-crepitant râles. Præcordial dulness increased; impulse strong; no thrill; apex behind ensiform cartilage. Over base double murmur; the systolic coarse, loud, maximum intensity at junction of third right costal cartilage, with sternum, heard in carotids, diffused over chest, especially on right side, also heard in right infra-spinous fossa. The diastolic murmur confined to region of base, heard over lower portion of sternum; replaces second sound. A feeble impulse and thrill felt at seat of maximum intensity of systolic murmur to right of sternum. Engorgement grew worse; some difficulty in swallowing; slight delirium, then drowsiness. Death quiet.

Duration of symptoms following rupture: Over 10 days; not over 3 weeks; exact time uncertain.

Autopsy: Lungs hyperæmic, œdematous. Pericardium entirely adherent to heart. Heart and valves showed nothing of importance. Aneurism as large as apple of moderate size, with opening into it from aorta about ½ inch above valves. Compressed right branch of pulmonary artery, right bronchus, and vena cava. Vena cava pressed forward and outward by it; occluded from entrance into auricle up to ½ inch below entrance of azygos vein. The opposite sides of intima of cava at its opening into auricle were partially adherent, probably congenitally, leaving one passage on each side; the one admitting a probe, the other the little finger. Three perforations existed from the sac into the cava; one the size of a three-penny piece, the others much smaller. Their edges were thin, jagged, and bordered by red areolæ on the lining membrane of the vein.

CASE XIII.—Reported by Jacoby. *Berlin. klin. Wochenschr.*, March 28, 1870, 158.

Previous history: Railway employee, draughtsman, always been well except hemidrosis of upper half body; did not know whether of lower half of body as well. First symptom a hindrance of motion in neck and some swelling there. Next day same.

Symptoms following the rupture: On this second day, as he leaned over his drawing, had sudden sense of being unwell; blueness and swelling of face and neck developing in course of two days. On third day intense blueness with œdema, and coldness of upper half body; sharp, zigzag color-line of demarcation at lower part of thorax; scleræ dark bluish-red; jugular veins prominent, very full. Sweat over whole upper part body. Radial pulses isochronous, small, compressible; left pulse much weaker than right. No dyspnœa at first, but sense of oppression. No pain. Percussion and auscultation of lungs normal. Heart's impulse feeble. No pulsation or thrill

in upper part chest. Loud buzzing murmur of greatest intensity at position for listening to aortic valves; loudest with systole, but continuous; and lasting, though not so loud, through diastole; widely diffused over chest. Not heard behind to left of vertebral column over the aorta. Not heard in femorals; faint in carotids. Besides murmur, two normal sounds could be heard at apex. Œdema increased; very little urine voided; dyspnœa developed, then orthopnœa. Some roughness of voice. Very somnolent. Death with symptoms of slow carbonic dioxide poisoning.

Duration of symptoms following rupture: 7 days.

Autopsy: Cyanosis largely disappeared; cutaneous ecchymoses both sides thorax; œdema persistent. Double hydrothorax. Nothing characteristic in lungs. Heart small; valves normal. Large saccular aneurism of ascending aorta, 9 cm. broad in widest part. Lining of sac atheromatous, uneven, and with numerous small aneurismal dilatations in the wall, of size of pea to that of walnut. Aneurism evidently an old one. Vena cava, at about its entrance into auricle, narrowed to size of crow's quill, and closely adherent to one of the little aneurisms of the size of cherry; and a slit-like perforation 4 lines long with smooth edges opened into this. A colorless old clot projected through it from the sac, and thus still more narrowed lumen of cava. Shortly above this very narrow part cava resumed normal size. Bronchi, œsophagus, and trachea not compressed.

CASE XIV.—Reported by Farrington. *Phila. Med. Times*, January 3, 1874, 216.

Previous history: Woman, domestic, aged sixty-one; moderate indulgence in alcohol; no venereal history; rheumatism seventeen years before, otherwise always well. About a year previously small tumor appeared right side of chest near sternum. No cause known unless it was repeated straining in lifting clothes-baskets. In about two months sharp pain down arm to elbow; sometimes in chest in region of tumor. These pains persisted. Voice hoarse for eighteen months No cough; no dyspnœa.

Symptoms following the rupture: After great excitement there developed intense cyanosis and great dyspnœa. Extremities cold; pulse small, rapid, short, sharp; no difference between the two sides. Pulsating tumor with dulness and thrill, extending 3 inches to right of sternum from second to fourth rib. Also double murmur, with greatest intensity over this region, transmitted into vessels of neck; diastolic portion loudest. Systolic murmur at apex also. Lungs normal. Symptoms persisted with great restlessness and an attack of vomiting.

Duration of symptoms following rupture: About 15 hours.

Autopsy: Aneurism, size of apple, from junction of ascending and transverse portions of arch, projecting forward and to the right. Vena cava compressed by aneurism, and in the wall of the vein, at junction of innominate veins, was a transverse slit, ½ inch in length, communicating with aneurism. Aorta showed chronic endarteritis. An aneurismal sac in descending aorta, pressing on upper lobe left lung. Heart not hypertrophied. Aortic valves thickened and slightly insufficient.

CASE XV.—Reported by Bonnarel. *Thèse de Paris*, 1875.

Previous history: Coachman, aged fifty-six; intemperate; always well until eighteen months previously, when an attack of dyspnæa necessitating rest from work for a time. Since this occasion respiration been somewhat oppressed.

Symptoms following the rupture: Without known cause dyspnœa greatly increased and cyanosis appeared. When seen after eight days exhibited orthopnœa; face, neck, upper extremities, and upper part trunk cyanosed, more on right than left side; small vessels distended, especially on nose and cheeks. Face and upper extremities [and thorax?] œdematous. No œdema of lower part trunk or lower limbs. No pain. Voice harsh. No venous pulsation in swollen jugular veins. Left radial pulse weaker than right. Apex beat lower than normal; no thrill. A strong, not harsh, systolic, blowing sound heard with maximum intensity at inner part first right intercostal space. Heard also over base; also posteriorly along spine. Respiration scarcely audible on right side. On left side sibilant râles. Dyspnœa and cyanosis increased; death.

Duration of symptoms following rupture: 16 days.

Autopsy: Entire arch aorta, particularly ascending and transverse parts, enormously dilated; 35 cm. its greatest diameter. Displaced vena cava and right phrenic nerve and the lungs somewhat, especially the left. Flattened right bronchus slightly. The dilated aorta presented several secondary aneurismal dilatations, one of them size of egg. Vena cava remarkably elongated; its walls intimately adherent to those of aorta. A slit-like perforation, 1 cm. long, opened into aorta about 13 cm. above the aortic valves. About 5 mm. above this was another perforation, round, 6 mm. in diameter, with ragged edges. Heart hypertrophied; valves normal. Some fluid in pleural cavities.

CASE XVI.—Reported by Schnaubert. *Ejened. klin. Gaz.*, St. Petersburg, 1881, i. 81, 105.

Previous history: Butler; aged forty-two; moderate drinker; never syphilis; fifteen years ago kicked in chest by horse, but soon recovered; three years ago asthma and palpitation, which soon disappeared.

Symptoms following the rupture: After great exertion at nocturnal conflagration had chill; then sense of heat in head, dysphagia, swelling of face and neck. Examination two days later showed head, neck, arms, and chest down to third rib bluish-red and considerably swollen. Veins distended, but not pulsating. Mucous membrane of mouth and throat purple and conjunctivæ injected. Some difficulty in swallowing. Vomited several times. Dulness on percussion, extending from middle of sternum to 3 cm. to right of its right edge and down to fourth rib. No pulsation in any part of chest. Loud, rasping, double murmur over area of dulness and over sternum; less distinct posteriorly over centre of left scapula. Radial pulses equal. Respiration rather stertorous; 24 in the minute. Some somnolence. Noises in the ears. Temperature in the axilla 36° C. Later, cyanosis and œdema reached to

lower ribs. Murmur was sometimes absent, sometimes only systolic or diastolic, sometimes double. Dulness extended somewhat. Hydrothorax developed. Œdema reached to hips and penis. A slight pulsation at times felt in region of subclavicular dulness. Dyspnœa; hallucinations; increasing cyanosis; delirium; death.

Duration of symptoms following rupture: 5 weeks.

Autopsy: Aneurism, size of hen's egg, starting about 5 cm. above aortic valves and extending toward right. Compressed right branch of pulmonary artery and the vena cava. Was adherent to right lung. Vena cava greatly distended; but about 5 cm. above the heart much narrower, compressed by aneurism, and filled with thrombus. In centre of compressed portion was a circular perforation, ½ cm. in diameter, opening into sac. Compact, adherent thrombus in right jugular. Right auricle and ventricle distended. Lungs œdematous. Bloody mucus in bronchial tubes. Hemorrhagic erosions in stomach.

CASE XVII.—Reported by Halla. *Zeitschr. f. Heilkunde*, 1882, iii. 122.

Previous history: Smith; aged sixty; always healthy; severe work.

Symptoms following the rupture: Suddenly, while lifting iron, developed cyanosis and œdema of upper half of body. Left arm swollen before the right. Jugular veins dilated; dense network of dilated veins on thorax; some dyspnœa; drowsiness; more or less dysphagia. When seen after sixteen days exhibited extreme cyanosis and œdema of upper half of body, with irregular line of demarcation in region of navel. Dense network of veins on chest. Eyes prominent. Some slight œdema of genitals and upper part thighs, and some dyspnœa and dysphagia. No tumor visible on chest. A diffuse, weak, systolic trembling without thrill felt from under right clavicle to right nipple; stronger than apex-beat. Very marked thrill on left side of neck, especially above left clavicle; weaker thrill on right side neck. A percussion dulness extending about 3 fingers' breadth from middle line toward sternal end right clavicle, and joining the cardiac dulness extending nearly to right nipple line. A very loud, sharp, blowing, systolic murmur heard with greatest intensity over aortic cartilage, widely diffused, prolonged into diastole. Two normal sounds at apex. Aortic and pulmonary second-sounds present. A very loud systolic murmur heard above and below left clavicle; weaker murmur in carotids and under right clavicle, and in last position was loudest during expiration. Left pulse slightly smaller than right; both show influence of respiration. Respiration anteriorly vesicular, weak on left side; posteriorly rough and broncho-vesicular except right upper part, where was vesicular. Tongue swollen; voice hoarse; paralysis of right posterior crico-arytenoid, with œdema of larynx. Later, double hydrothorax; catarrhal and œdematous swelling of bronchial mucous membrane; increasing œdema, dilatation of veins, dyspnœa, dysphagia, and cyanosis; expectoration tinged with blood; chemosis of conjunctiva of right eye; increasing laryngeal œdema and stridor; oozing of serum from skin. Temperature had been subnormal; urine scanty, no albumin. Death after short sopor.

Duration of symptoms following rupture: 30 days.

Autopsy: Aneurism of aorta, larger than fist, beginning 3 cm. above valves and reaching to innominate artery, projecting to right; interior atheromatous. Contained several small dilatations. Vena cava behind and to right of aneurism; its whole length, except lower 2 cm., as well as about 3 cm. of left innominate vein adherent to sac. Cava compressed, the greatest narrowing being just below entrance of azygos vein. 1 cm. above azygos was a slit-like perforation, 7 mm. long, with smooth, rounded edges, opening into aorta. At this point cava began to widen again.

CASE XVIII.—Reported by Damaschino and Lavin. *La France Médicale*, 1882, i. 805, 819.

Previous history: House-painter; aged fifty; always healthy. Twenty-two years before had strained himself lifting; felt sense of oppression in chest for some hours, but always well since.

Symptoms following the rupture: Suddenly, after stooping and lifting weight of 150 pounds, his friends noticed that his face was blue. Was no pain, oppression, or palpitation. Cyanosis spread to whole of head in a few hours. By next morning, face, neck, and right arm swollen. By afternoon, left arm also, and he felt constriction in throat, and general feebleness. By evening, all upper part body exhibited venules. In five days a little roughness of voice, and veins of face, arms, trunk, including subcutaneous abdominal veins, commenced to develop. When seen six days after onset there was cyanosis of skin and mucous membrane, œdema of upper portion of body, distention of veins, and development of network of venules. Both arms very œdematous, especially right. Upper part thorax slightly prominent, especially right side. Apex-beat feeble in sixth interspace close to sternum. Cardiac dulness seemed enlarged, but œdema prevented accurate determination. On right side a dulness from middle of clavicle to fourth interspace, extending about 3 fingers' breadth to right beyond sternum. A thrill at upper part right side, reaching above clavicle. A centre of pulsation in third interspace at right edge of sternum and a second centre at apex, not so well marked. In second interspace to right of sternum a rough, systolic murmur and a blowing, diastolic murmur, the two being almost, but not quite, continuous. 3½ cm. below middle of right clavicle was a very intense and actually continuous murmur, with systolic reinforcement. It was remarkable, especially during the reinforcement, for the peculiar sonorous and vibratory character. It was transmitted with the same characters to the base of the neck and even to the lower part of the thorax posteriorly on the right side. It disappeared at the right border of the vertebral column, and here could be heard in contrast the double aortic murmur first described, with the same characters as heard at the centre for this murmur at the base. Pulse regular. A double murmur heard in femorals. Axillary temperature normal, though the patient complained of feeling cold. Lungs normal. Cough and some bloody expectoration. No palpitation or pain. Some vertigo and oppression on walking. Patient improved, and after being under

observation nearly one month the œdema disappeared entirely from the face, arms, and neck, and almost from the trunk, and there was but very faint cyanosis when at rest, though it returned on quick motion. All the veins of trunk, upper extremities, and roots of thighs very greatly developed.

Duration of symptoms following rupture: Patient left hospital practically well, in the condition described, with collateral circulation well established.

CASE XIX.—Reported by Glasgow. *St. Louis Cour. of Med.*, 1884, xi. 407, and 1885, xiii. 1.

Previous history: Physician; aged fifty-seven; previous good health, except for several accidents. For some months, dyspnœa on unusual exertion.

Symptoms following the rupture: On lifting a man he felt sudden rush of blood to upper part of body. Face and hands swollen and purplish. No dyspnœa or pain. Soon entire upper part body swollen, œdematous, and cyanosed. General enlargement of superficial veins, with spots of ecchymoses. Eyes half closed; pulse soft, full, jerking like that of aortic regurgitations. Dulness over sternum for 3 inches in breadth, and from second to fourth ribs. Strong heaving impulse here, with exquisite thrill, especially at right edge of sternum. Double murmur heard at aortic cartilage and transmitted over sternum and præcordium; diastolic portion ended musically. Over rest of chest only single systolic murmur could be heard; this audible in larger arteries, even in brachial. Appreciable pulsation in liver, and distention of large superficial veins of abdomen. A second examination, after somewhat over a month, showed remarkable improvement in everything except an increased percussion dulness and occasional great swelling of legs. Soon after this great dyspnœa on exercise began to develop, hydrothorax and general œdema appeared, dulness of aneurism increased greatly, but impulse could only be felt in third interspace to left of sternum. Systolic bruit faint; respiration feeble. Finally, there was increasing œdema, greatest on left side face, neck, and chest; marked cyanosis and dyspnœa. Death.

Duration of symptoms following rupture: 7 months.

Autopsy: Lungs engorged; large amount of fluid in pleural cavities. Heart enlarged; right auricle enormously dilated; left ventricle hypertrophied; valves healthy. Globular aneurism, 4¾ inches in diameter, occupying first portion of arch, extending to innominate artery. Compressed left innominate vein and pulmonary artery. Walls of aneurism atheromatous. A perforation, size of goose-quill, into vena cava situated at upper right side of sac; closed by a thick plate of laminated fibrin. Superior cava greatly dilated; the opening into aneurism immediately below entrance of innominate veins. Azygos vein greatly enlarged. Inferior cava greatly distended.

CASE XX.—Reported by Wetterdal and Wallgren. *Upsala Läkarenförenings Förhandlingar*, 1884–5, xx. 325.

Previous history: Man; aged thirty-four; always done hardest work without difficulty. Probably had had syphilis, but not certain. Used alcohol to excess.

Symptoms following the rupture: Suddenly, while putting forth violent

exertion, had a chill; felt a sense as of something sticking in throat; general sense of soreness in thorax; headache. In half hour blueness in face and hands, and dizziness. On reception into clinic that afternoon patient complained of dyspnœa and severe headache and dizziness on sitting up. Face, arms, and upper part trunk very cyanotic, with a well-defined line of demarcation at lower part thorax. The larger veins much swollen. Great œdema of the cyanotic portions, with distention of the veins. Cardiac dulness not enlarged. No abnormal dulness in chest, and no heart-murmurs or abnormal pulsation. No pulsation in jugular veins. Above right sterno-clavicular articulation and toward right external jugular vein was a booming sound with cardiac systole. Radial pulses isochronous. Dyspnœa, headache, and œdema rapidly increased. Ten hours after admission gave sudden cry and died.

Duration of symptoms following rupture: Less than 24 hours.

Autopsy: Sacculated, fist-sized aneurism of ascending aorta, closely adherent to superior cava. In vena cava 4 cm. above heart was a perforation into aneurism 7–8 mm. in diameter; its edges sharp, a little swollen, and inverted toward vein. Lungs œdematous.

CASE XXI.—Reported by Turner. *Transactions Path. Soc. London*, 1885, xxxvi. 148.

Previous history: Man, aged forty. History of venereal disease; always hard work; no sudden strain or assignable cause.

Symptoms following the rupture: Disease began with dyspnœa. When seen after seven weeks there was dyspnœa; much swelling of upper parts; face dusky; superficial veins enlarged, especially over chest. Loud to-and-fro *bruit* over aortic valve and apex; obscure pulsation over upper part sternum; evidence of pressure on right bronchus; some huskiness of voice and contraction of right pupil. Death from increasing pulmonary obstruction.

Duration of symptoms following rupture: 23 weeks.

Autopsy: The ascending aorta much elongated, with a large sacculated aneurism projecting toward the right. Probably was a dissecting aneurism originating in a transverse rupture of the vessel. Mouth of aneurism on convexity of vessel extending from ¾ of an inch above free border of aortic curtains upward 2–3 inches. Sac had compressed, closed, and finally passed through the vena cava, completely separating its proximal and distal portions and projecting between them. Distal portion looked like a short arterial trunk given off from aneurism near its upper border. There was free passage of blood from sac into vein. Aorta quite atheromatous. Aortic valves calcareous; orifice stenosed. Mitral valves thickened. End of trachea and right bronchus compressed by aneurism. [Right?] lung congested, œdematous, and adherent to chest-wall. Much effusion in left pleural cavity. [Proximal portion of vena cava probably pervious, as it is not stated to the contratry.)

CASE XXII.—Reported by Shannon. Report of Surgeon-General of Army (U. S.), 1886, p. 101.

Previous history: Sergeant. No history of previous trouble, except that for few days had had slight dizziness on stooping.

Symptoms following the rupture: At once, after great excitement, felt a terrible rushing of blood to head. Vision grew dim, strength failed, had to be assisted to room. Could talk. Face very blue and swollen; ears especially cyanosed; eyes protuberant, with bloody tears; varicose, greatly distended and pulsating jugulars. Respiration only a little accelerated and noisy; constant effort to clear nostrils; voice like one with bad cold in head. Neck swollen, finally extremely engorged. No blueness or swelling of arms. Vomiting at times. Heart regular, normal. A murmur at second right costal cartilage with second sound; heard at first examination, but repeated auscultation afterward failed to find it again. Respiration somewhat bronchial and noisy. Pulse full, soft. No unconsciousness or paralysis at any time. Not even stupid until near end. Thirst constant. About two hours after first commencement had severe pain between scapulæ lasting until death. Patient lay down; later effort to rise produced some dyspnœa. All symptoms next increased in intensity. Finally, terrible attack laryngeal dyspnœa ended in death.

Duration of symptoms following rupture: 6 hours.

Autopsy: Cyanosis of face disappeared shortly after death. Lungs congested. Old adhesions of limited extent of pericardium to aorta. No pleural adhesions. A true aneurism of aorta extended from just above coronary arteries to innominate artery, where it became fusiform, and continued in transverse aorta to and including the opening of left subclavian artery. Aneurism 8 inches long on total convexity, 3 inches deep, 3½ inches wide, involved chiefly right upper posterior part of vessel. In posterior wall a small lacerated opening apparently recently torn, leading into distal portion of vena cava close to innominate veins. Proximal portion of vein obliterated in sac-wall, except ½ inch nearest to heart, which was patulous but narrowed. Left superior intercostal size of either innominate vein. No œdema of glottis found. Heart normal.

CASE XXIII.—Reported by Christianu. *Spitalul. Bucuresci.*, 1887, vii. 74.

Previous history: Saloon-keeper, aged sixty-one; intemperate; never syphilitic; previously always well. For some time cough, which became worse two weeks before seen; occurred in paroxysms which threatened suffocation; of late accompanied by severe pain in chest and swelling of neck. Examination showed some cyanosis and swelling of face, neck, thorax, and upper extremities. Swelling elastic rather than like œdema. Livid patches containing distended veinules. No murmurs; pulse strong, not accelerated. Respiration not accelerated; no dyspnœa. Dulness under right clavicle to third interspace and from middle line of sternum to 4–5 cm. to right of right sternal border. Some subcrepitant râles in lower part lungs. After three weeks somewhat improved and left hospital.

Symptoms following the rupture: Returned in two days with symptoms much aggravated and with orthopnœa. In one week more systolic murmur present, best heard in the region of the aorta, and in three days more both systolic and diastolic murmur, best heard in right supra-spinous fossa. Tremor of chest-wall, with thrill in third and fourth interspaces in front. Cyanosis, œdema,

and dyspnœa increased and became extreme. Eyes projected and were bloody. Systolic murmur became so loud that could be heard on simply approaching ear to chest. Finally, restlessness, delirium, cyanôsis of whole body, stertorous respiration, great œdema of nostrils, death by suffocation.

Duration of symptoms following rupture: 16 to 18 days.

Autopsy: Heart hypertrophied. Aorta and large vessels atheromatous. Aneurismal dilatation size of goose-egg from origin aorta up to transverse portion of arch. Vena cava adherent to sac, compressed, and a small perforation 1 cm. in diameter, with irregular edges, connected the vein and the aneurism. Azygos vein much dilated. Lungs, liver, spleen, and kidneys congested.

The exact time of the rupture in this case is not clear. If it occurred two weeks before the patient was first seen, the duration of the acute symptoms would be about seven and a half weeks. The fact that improvement occurred, and especially the possession of irregular edges by the perforation, as though it had been recently formed, indicate that the rupture most probably took place at the time the great aggravation of symptoms with the development of orthopnœa occurred.

CASE XXIV.—Reported by Gulliver. *Transac. Path. Soc. London*, xxxviii. 1887, 120.

Previous history.
Symptoms following the rupture.
Duration of symptoms following rupture.
[This was simply a card pathological specimen, unattended by clinical notes.]

Autopsy: Arch of aorta much dilated and diseased. At top of ascending portion was a circumscribed aneurismal dilatation which communicated with superior vena cava by a circular perforation size of goose-quill. Left internal jugular entirely closed by a clot, and the innominate at its termination had its lumen entirely obliterated by the pressure of the aneurism; and its opening into the vena cava was quite invisible. Cava elongated, being stretched by the aneurism.

CASE XXV.—Reported by Arkle and Bradford. *Brit. Med. Journ.*, 1888, ii. 1387; *Transac. Clin. Soc.*, London, 1889, xxii. 69.

Previous history: Shoemaker, aged sixty-one; never rheumatism or syphilis; winter cough for last six years; always well in other respects. Twenty months previously began to have pain in right mammary region of a dull, aching character; also dyspnœa, worse on the least excitement.

Symptoms following the rupture: About nine days before entering hospital seized with pain and swelling in neck. In a few days considerable swelling of right arm. On admission was an almost black cyanosis of face, most marked on right side. Head and neck œdematous on right side, but little on left. Right arm greatly swollen and pitted. Chest slightly œdematous, especially on right side. Left side chest and left arm but little swollen. No œdema of lower portion of body. Some dilated veins along borders of ster-

num, and a ring of them horizontally around base of thorax. Respiration labored, accelerated. Paroxysms of coughing in which cyanosis was much increased. Some impairment of resonance in right infra-clavicular region in second and third interspaces, reaching the edge of the sternum. Visible pulsation in these interspaces. Numerous bubbling râles over chest. A thrill over the dull area, and a loud murmur. This murmur peculiarly harsh, continuous; loudest in systole; gradually diminishing in diastole; audible all over right infra-clavicular region, but maximum intensity in second right interspace some 2 inches from sternum. Apex in sixth interspace outside nipple line; systolic murmur here not conducted into axilla. Aortic second sound inaudible at base and apex. Cyanosis increased. Died suddenly.

Duration of symptoms following rupture: About 10 days.

Autopsy: A sacculated aneurism involved whole of ascending and greater part of transverse portion of arch; projected upward and to right; was flattened from before backward. Sac displaced right lung and reached surface of chest in second and third right interspaces. Superior vena cava lay behind aneurism and was compressed by it. About 1½ inches from commencement of vein, just above spot where it passed through pericardium, was a perforation into aneurism; diamond-shaped; ¼ inch in diameter, with distinctly rounded edges. No appearance of any recent tear or rupture of any kind. Aortic valves insufficient; other valves normal. 1½ pints fluid in right pleural cavity, rather less in left.

CASE XXVI.—Reported by Kraus. *Prag. med. Wochenschr.*, 1888, 119, 130.

Previous history: Revenue officer, aged fifty-two; sixteen months previously began to suffer from cardiac asthma, with dyspnœa and pain in region of heart and in left arm. In following month noticed that neck and region of chin were swollen, and felt as though mucous membrane of throat was swollen. Respiration was through all this decidedly, though varyingly interfered with, though the attacks of asthma were less prominent. Gradually veins of neck swelled, and cyanosis of face developed. Six weeks before symptoms of rupture appeared the chest became œdematous and exhibited bluish spots and distended veins.

Symptoms following the rupture: Suddenly there occurred very severe attack of dyspnœa amounting to orthopnœa; palpitation of heart; præcordial anxiety; very deep cyanosis. Was brought on same day to hospital in this state. While under observation exhibited rapid, shallow, noiseless respiration, with attacks of dyspnœa; pulse small and rapid, but slower during asthmatic attacks, alike in both radials. On face, neck, and upper part thorax cyanosis, elastic œdema, and dilatation of veins. Eyes prominent. Noise in ears. Left arm œdematous, and later right arm also; hand and fingers not cyanotic. Lower half body not affected; no sharp line of demarcation. An indistinct pulsation to right of sternum from second to fifth rib; no distinct dulness here; a thrill from third to fifth rib on right side, which later disappeared and then returned. Apex beat could not be distinctly

located. Rough blowing systolic murmur, with maximum intensity in second right intercostal space; rather widely diffused, and heard in carotids. Examination of lungs negative, except some dulness in lower portion with faint bronchial breathing here and a few râles. Cyanosis, œdema, and attacks of dyspnœa varied, but finally grew worse. Œdema of larynx; of genitals; and slightly of upper parts of thighs. Sopor; hallucinations; unconsciousness; death.

Duration of symptoms following rupture: 40 days.

Autopsy: About 2 quarts fluid in each pleural cavity. Lungs congested; œdematous; lower portion atalectatic. Mitral and aortic valves a little thickened; lateral leaflets of latter adherent to each other. Dilatation of whole ascending aorta beginning a finger's breadth above valves. 3 cm. above sinus of Valsalva of posterior leaflet was a secondary sacculated aneurism size of walnut; projecting from posterior and right side; in intimate contact with root of right lung, and displacing vena cava to right. Vena cava fused with sac and entirely obliterated for 4½ cm. above heart; above this much dilated. About opposite entrance of greatly dilated azygos vein were two smooth-walled perforations from cava into sac; the one the size of a pea; the other that of a hemp-seed.

The author believes that the rupture took place some time before the acute symptoms developed, and claims that the character of the perforation shows this beyond doubt. Other cases which we have abstracted prove, however, that the smoothing of the edges of a perforation takes place very rapidly, and that there is consequently no reason to believe that the rupture did not occur at the time at which the sudden increase of the symptoms would indicate.

CASE XXVII.—Reported by Sisley. *Lancet*, 1889, i. 1184. See also note by Ewart. *Lancet*, 1889, ii. 312.

Previous history: Laborer, aged thirty-five; temperate; scar on penis; rock fever at Gibraltar; no history of accident. For some months cough, but no shortness of breath. Eyelids were puffy and face somewhat swollen on returning from work in evenings. This condition only seen at night, and had always disappeared by morning.

Symptoms following the rupture: On one occasion went to work, but, not feeling as well as usual, went into hospital for advice; though not considering himself seriously ill. Face and neck were deeply cyanosed; eyelids puffy and ears almost black. Arms slightly cyanosed; rest of body natural color. No pain; no paralysis. Mental state clear, but was drowsy. Pulse full, regular. Respiration not noisy or accelerated. Double murmur over middle of sternum; nothing else abnormal on physical examination of chest. After venesection a venous pulse was observed in jugulars and cephalic veins. Face and arms became gradually more cyanosed; respiration difficult and noisy, with expiratory stertor. Became more drowsy, and finally comatose. Died about two hours after admission.

Duration of symptoms following rupture: Less than 12 hours.

Autopsy: Saccular aneurism ¾ inch above posterior leaflet of aortic valve. Sac measured about 1½ inches in transverse diameter, and 2 inches from above downward. Was in contact with vena cava and right auricle. Vena cava quite patulous. Immediately above its entrance into right auricle was an opening into aneurism. This of irregular form, a little smaller than a sixpence, and with ragged edges projecting slightly into vein. Aortic valves not thickened. Right ventricle slightly hypertrophied. Lungs congested.

CASE XXVIII.—Reported by Gairdner. *Lancet*, 1889, i. 1233.

Previous history: Laborer, aged forty-four. For two years been conscious of slight degree of pulsation in neighborhood right sterno-clavicular region, which caused him no concern.

Symptoms following the rupture: Experienced a sensation as of something giving away near heart. No pain, but faintness and cold sweat lasting ten minutes; never recurred. Swelling of face and right arm followed, but had considerably diminished in degree when first seen after one week. At that time exhibited very marked cyanosis; general anasarcous swelling of upper half body; some dilatation of small superficial veins all over front, especially in lower sternal region, extending thence to præcordial, right hypochondrial and right manubrial regions. Veins of hands distended, especially on left side. Very loud and characteristic murmur heard all over front of chest, but having mainly the distribution of aortic double murmur. The ventricular-systolic element loudest at base, and diastolic element at least relatively loudest toward lower sternum. Some displacement of præcordial dulness downward and toward the left. Another and distinctly abnormal dulness, easily demarcated from the præcordial, occupied entire manubrium of sternum, and extended both to right and left; but its limits in former direction were difficult to state, owing to anasarca. Appreciable, but not very definite, pulsation in jugular fossa. A faint tracheal quality in respiratory murmur, as heard at upper sternum. Pulse rapid, decidedly stronger in right wrist than in left; in latter not abnormal, but in former had character of unfilled arteries. Pupils normal. But little cough. No signs of laryngeal implication. Respiration without difficulty at first; finally stertorous, but was no dyspnœa.

Duration of symptoms following rupture: 15 days.

Autopsy: Transverse portion of arch of aorta generally dilated, but with two more localized aneurismal dilatations; one extending to right, other to left. The first of these compressed superior vena cava and was adherent to it, forming a round bulging into interior of vein, corresponding to middle and upper third of this vessel. On summit of bulging a rounded perforation, ¼ inch in diameter and 1¾ inches below origin of innominate vein.

CASE XXIX.—Reported by Pepper and Griffith in this communication.

A review of the salient features of these cases and of the one now reported shows that in many instances the diagnosis is easily possible

during life, while in others it is perhaps impossible. A correct diagnosis was, in fact, made by Mayne and by Glasgow in their respective cases (VII. and XIX.). Gull, in the case reported by Eastes (X.), recognized an abnormal intra-thoracic venous communication, perhaps with an aneurism; and Gallard, in his first case (XI.), as well as in that reported by Bonnarel (XV.), diagnosed an aneurism of the aorta, and discussed the probability of there being a communication between it and the superior vena cava. Damaschino and Lavin made, with apparent reason, the diagnosis of the affection in their case of recovery (XVIII.).

The recognition of the presence of varicose aneurism of the aorta and superior vena cava may be based upon the following principal symptoms, deduced from a study of the different reported cases:

1. *Cyanosis, œdema, coldness, and distention of the veins of the upper part of the body, with other evidences of obstruction to the circulation of blood in the tributaries of the superior cava.*—These symptoms have been present to a greater or less degree in all the reported cases of which clinical histories are given, and their diagnostic indications are evident. No lesion could produce such a condition, with its peculiar localization, except some great obstruction to the course of the blood in the superior cava. The thorax, however, does not always share the lividity and œdema of the head and arms; this variation depending solely on the position of the perforation or of the compression. If, namely, the obstruction in the cava is situated below the point of entrance of the azygos vein, the chest will share in the venous congestion; if, on the other hand, the obstruction is above the azygos vein, the removal of blood from the chest-wall is not interfered with, and no œdema develops. In certain cases the reason for the absence of thoracic œdema is not entirely clear, and often depends on the lack in the published report of a sufficiently detailed statement regarding the exact position of the obstruction in the cava. When the thorax is involved there is sometimes a sharp line of demarcation between the upper affected and the lower unaffected portion of the body, marked by the numerous small varicose veins around the lower part of the thorax. It has happened more than once that a patient has shown a greater degree of œdema on one side than on the other. This condition may be produced in several ways. For example, in the case of Arkle and Bradford's (XXV.) a long-standing

obliteration of the left innominate vein had allowed the establishment of the collateral circulation, so that the sudden rupture of the aneurism produced œdema almost limited to the right side. A similar unilateral œdema and cyanosis of the upper portion of the body would be produced by communication of an aneurism with the left innominate vein, as seen in the case reported by Chabond (*Lyon Méd.*, 1873, No. 26; *Virchow-Hirsch Jahresb.*, 1873, ii. 147). Another case illustrating differences in the swelling of the two arms is that of Cossy (VI.). In this the opening into the aneurism from the vena cava was situated close to the bifurcation of the cava, in such a position that the blood-current was directed into the right innominate vein, producing consequently much greater swelling of the right arm. In some other instances the causes of the slight differences observed are not apparent. When death has very rapidly followed the rupture of the aneurism there may have been no time for œdema and lividity of the arms to develop. This was the condition in the case reported by Shannon (XXII.), in which only the face and neck gave evidence of the change. The onset of the symptoms in this instance was extremely sudden, and death occurred in six hours.

The cyanosis is probably not due to the admixture of arterial and venous blood; otherwise we should see it well marked in cases of rupture of an aneurism into the pulmonary artery. It depends simply on obstruction from compression by the aneurism, and from the backward pressure of the arterial blood into the vein. In the patient of Christianu's (XXIII.) the whole body finally became cyanosed.

Slight œdema has in some instances been witnessed in parts of the lower portion of the body, though always secondary to that above. Thus, in Thurman's patient (II.) there was slight œdema of the ankles and scrotum, and in Schnaubert's (XVI.), Halla's (XVII.), and Kraus's (XXVI.), of the genitals, hips, and upper parts of the thighs. In the case of Glasgow's (XIX.) there finally developed general œdema. Œdema of the lower portion of the body in these cases is due, according to Halla, to the overfilling of the inferior cava through the necessity which it is under of receiving a large part of the blood belonging to the superior cava. This same passive congestion explains the scanty secretion of urine which has been noted in a number of instances. In a considerable number of cases large amounts of fluid have been found in both pleural cavities, while in other instances

there was none. The difference undoubtedly depends largely on whether or not the circulation in the azygos vein is interfered with.

Besides the cyanosis and œdema, dyspnœa may be present, either from the outset or later in the disease. In our own case it was intense, as it was in a large number of others. In others, again, it was absent, or slight, or not referred to by the writer. Thus, in Cossy's patient (VI.) it was slight, and in Gairdner's (XXVIII.) nearly absent, and it does not appear to have been well marked in the case of Hayden's (XII.). It is not at all essential that dyspnœa be present, as it is in the event of rupture of an aneurism into the pulmonary artery. Its development depends on various factors, such as double hydrothorax with consequent compression of the lungs; œdematous swelling of the mucous membrane of the nose, pharynx, trachea, larynx, and bronchi; œdema of the lungs; pressure by the tumor on the respiratory tube; overfilling of and consequent irritation of the nerve-centres with venous blood. In Shannon's case (XXII.) death was directly due to an attack of extreme laryngeal dyspnœa.

Still other symptoms indicating venous obstruction may be seen. Cough is a frequent and natural attendant on the dyspnœa, and râles of various sorts are often heard. There was sometimes expectoration, and not infrequently it was tinged with blood, as, for example, in the case of Thurman's (II.), Halla's (XVII.), Schnaubert's (XVI.), and of Damaschino and Lavin's (XVIII.). In our own case a considerable pulmonary hemorrhage took place, due, probably, to an erosion of the trachea by the aneurism. Thurman claims that the dyspnœa, cough, and other evidences of pulmonary congestion, witnessed during life or after death, are due to the circulation through the lungs of an admixture of arterial and venous blood, and the resulting irritation by it of the bronchial mucous membrane.

In some cases there has been difficulty in swallowing, probably due either to pressure of the aneurism on the œsophagus, or, in other instances, doubtless to the excessive distention of the tissues of the neck with serum. Bloody tears were seen in Shannon's patient (XXII.), and blood oozed from the conjunctivæ in our own case, and in that of Christianu's (XXIII.). A not infrequent symptom as the end approaches is a marked tendency to sleepiness, and, finally, coma. In some cases delirium was present before the coma developed. Attacks of faintness have also been reported, as in the case of Goupil's (VIII.).

In a few instances (IV., IX., XIV., XXIII.) excessive restlessness was noted. Headache, too, is naturally present at times. In Kraus's case (XXVI.) and in that of Schnaubert (XVI.) there was noise in the ears, and in Cossy's patient (VI.) annoying and persistent noise in the right temple and ear, a sense of painful tension in the head, and giddiness. Giddiness was observed in a number of other cases. Convulsions were uncommon, though they occurred shortly before death in Mayne's patient (VII.). Pain in the chest was sometimes observed, and in some instances was among the first symptoms appearing. Coldness of the upper portion of the body is a very common symptom. The temperature chart of our own patient is an illustration of this fact.

2. *The suddenness of onset of the symptoms.*—With the recognition of impeded return of blood through the superior cava the diagnosis is by no means made. Obstruction to the flow of blood in the cava from various other causes may, as already stated, produce the same symptoms. Still, the comparative suddenness of their development in cases of varicose aneurism constitutes a most valuable diagnostic sign. This rapid development was seen in all the cases in which the mode of onset is reported. Sometimes it was exceedingly abrupt. For example, in the case of Shannon's (XXII.) the symptoms were intense, and immediately followed a degree of excitement. Young's (IV.), Tripier's (IX.), and Gallard's (XI.) patients were suddenly taken during the night with very severe symptoms of the rupture. The patient of Law's (V.) became cyanosed and swollen without any premonition while lying down after a morning walk. Cossy's patient (VI.) woke in the morning with evidences of the rupture; Mayne's (VII.) was stooping; Jacoby's (XIII.) was leaning over his drawing; Farrington's (XIV.) had just been brought from the clinic in a state of great excitement; Halla's (XVII.), Damaschino and Lavin's (XVIII.), and Glasgow's (XIX.) were lifting heavy weights; Wetterdal and Wallgren's (XX.) was also putting forth violent exertion. In certain other cases no history of at least such a very sudden and alarming beginning is given, and sometimes it clearly was absent. Thus, in the case reported by Sisley (XXVII.), though the onset seemed to have been sudden, the patient was not aware of any great change in his condition. How long a time was actually required for the symptoms to develop in Eastes's case (X.) is not stated, nor is it en-

tirely clear in the cases of Christianu's (XXIII.) and Kraus's (XXVI.). In Thurman's patient (II.) several days seem to have been needed for the swelling and cyanosis of the face to become apparent, and the same is true of Arkle and Bradford's patient (XXV.). In Hayden's case (XII.) it is not stated that there was any sudden onset, so far, at least, as known to the patient. In our own case it could not be ascertained with just what rapidity the symptoms arose, but it is certain that they were not long in developing.

It is quite evident from a study of these cases that there is no instance reported in which there is an account of simply a very gradual increase in the gravity of a previously existing condition. Only a few days at the most were necessary for the characteristic symptoms to appear.

The rapid development of symptoms is not, however, absolutely pathognomonic of rupture of an aneurism into the vena cava, although it renders the fact of its occurrence extremely probable; for, although simple compression of the cava is usually slow in its onset, the symptoms may exceptionally make themselves known with comparative rapidity. A case is reported by Dujardin-Beaumetz (*Gaz. Heb. de Méd.*, 1879, 2 S., xvi. 19–23), in which extreme venous congestion was produced in the course of ten days, the cause being compression of the superior vena cava by an aneurism, and the development of a clot in the greatly dilated azygos vein, in which the collateral circulation had previously been carried on. He admits that he has been unable to find any other case recorded in which the symptoms came on so rapidly. There is an instance, however, reported by Duchek (*Prag. Vierteljsch. f. Prak. Heilk.*, 1854, xli. Also *Handb. spec. Path. u. Therap.*, 1862), in which rapid development of the symptoms of obstruction was probably due to the sudden production of a thrombus in the cava. The rapid formation of a dissecting aneurism could also by compression quickly produce symptoms of venous obstruction.

In many cases symptoms indicating a degree of obstruction had existed for months or years before the rupture took place. Sisley's patient (XXVII.), for example, had had for some months swelling of the face, which came on after the day's work; and in Mayne's case (VII.) swelling of the eyelids, face, and hands, and dyspnœa, were produced by working. Gairdner's patient (XXVIII.) had been conscious for two years of pulsation in the right side of the chest. In

Kraus's patient (XXVI.) there had been for sixteen months very evident and persistent œdema of the neck and throat, and later cyanosis as well. Dyspnœa, cough, and palpitation, and sometimes pain, had been noticed in a large number of cases. In Farrington's patient (XIV.) there had been for a year a small tumor in the right chest, presumably an aneurism. In our own case the previous existence of dilated veins upon the thorax was the only indication of the earlier presence of the aneurism. The existence of such symptoms, followed by a rapid and great increase in their severity, or by the accession of new symptoms, would indicate that some great and sudden change had taken place in the condition of the thoracic viscera—the most probable being, of course, the rupture of an aneurism. Many cases begin without previous symptoms. These would equally point to some great change in the thoracic contents.

3. *Evidence from physical examination of the presence of a tumor in the thorax, and the probability that this is aneurismal.*—No reference is had here to the murmurs found, as these will be discussed separately. If the presence of an aneurism is confirmed in this way, in addition to the indications already described, the diagnosis of rupture into the superior cava becomes greatly strengthened. In our own case the physical signs discovered in the chest were for the most part inconstant and contradictory, owing to the interference presented by the great œdema of the thoracic walls. One symptom, however, was nearly always observed, viz.: a degree of dulness below the right clavicle and under the upper portion of the sternum. A review of the reported cases shows that a percussion dulness, occupying this same region, was frequently present. Not uncommonly this centre of dulness exhibited a thrill, usually systolic in time, though in one case (X.) diastolic. Thrill could sometimes be detected elsewhere, also. Thus, in Mayne's and Cossy's cases (VII. and VI.) a purring tremor could be felt in the internal jugular and subclavian veins on the right side. A pulsation was present over the centre of dulness in many instances, but absent in more. Mayne (VII.) and Glasgow (XIX.) describe it as strong and heaving, and in most of the cases where it could be felt at all it was well marked, and oftener stronger than the cardiac impulse. In some cases, as in those of Hayden's (XII.), Turner's (XXI.), Kraus's (XXVI.), and Schnaubert's (XVI.), it was indistinct or feeble. In Farrington's patient (XIV.) there was a distinct

pulsating tumor visible to the right of the sternum from the second to the fourth rib. In our own case neither thrill nor pulsation could be felt. Mayne (VII.) and Glasgow (XIX.) describe the pulse as jerking, like that of aortic regurgitation, and consider it an additional proof of the existence of an aneurismal tumor. Gairdner (XXVIII.), also, refers to the pulse as that of unfilled arteries. In a number of instances it is reported as small and feeble, but there appears to have been nothing characteristic about it. It is sometimes stated to have been stronger in one wrist than in the other. Undoubtedly the difference has depended upon the varying degree of œdema of the arms, or some such factor, since aneurisms of the ascending portion of the arch would not produce this inequality. A venous pulse, as seen in the cases of Cossy's (VI.) and of Sisley's (XXVII.), is a very valuable indication of the presence of a varicose aneurism. The existence in any case of such well-recognized symptoms of aneurism as dulness on percussion, thrill, and pulsation, together with the symptoms previously described, render the diagnosis very probable.

There remains to consider the last of the evidences of varicose aneurism:

4. *The existence of a murmur characteristic of a communication between an artery and a vein.*—When this murmur is present the existence of a varicose aneurism becomes practically a certainty. Thurman first explained the nature of this murmur as occurring in spontaneous varicose aneurisms of the aorta, and dwelt upon its diagnostic importance, though it was not present in the case of aneurism of the aorta and superior vena cava reported by him (II. of this series, IV. of his own). Its extremely loud and distinct character is produed by the small size of the opening. The chief feature of the murmur is that it is *continuous*, this being due to the fact that the passage of blood from the aorta into the cava is a continuous one. During the systole the pressure is at its height, and the sound is consequently loudest and highest pitched. During the diastole the current from the artery to the vein depends on the elasticity of the arterial system acting upon the contained blood. The murmur is, therefore, still audible, though continually growing fainter and lower pitched until the next systolic intensification. A perfectly typical murmur of this nature was present in our own case. It was also observed in the cases of Eastes's (X.), Jacoby's (XIII.), Arkle and Bradford's (XXV.), and

Damaschino and Lavin's (XVIII.). In the first it is described as a soft, whizzing, very distinct murmur of a venous character, both diastolic and systolic, with its maximum intensity at the third right costal cartilage. It constituted a continuous churning sound, like that heard in the enlarged vessels of a thyroid gland. In Jacoby's case there was a loud, continuous buzzing murmur, loudest with the systole, and heard best at the second right costal cartilage. In the case reported by Arkle and Bradford the continuous murmur was peculiarly harsh, loudest in the systole, and heard under the right clavicle, though the maximum intensity was in the second right interspace about 2 inches from the sternum. Finally, the diagnosis made by Damaschino and Lavin depended largely on the perfectly continuous murmur, heard loudest 3½ cm. below the middle of the right clavicle. It was very intense, had a systolic reinforcement, and was remarkable, especially during the systole, for its peculiar sonorous and vibrating character.

A continuous murmur would seem almost necessarily to owe its existence to a varicose aneurism. Neither a simple aneurism nor any form of valvular disease could produce the continuous sound, since there would need be a short interval between the systolic and diastolic portion. In our own case the peculiar continuousness of the murmur was noted, as well as the fact that its quality was distinctly venous. The effort was made to explain it on the ground that there existed an aneurism of the aorta pressing upon the cava and producing a murmur in it; and that during the cardiac systole the aneurism expanded and the pressure became greater. Consequently, the pitch of the murmur was elevated, since the lumen of the vein was made more narrow. This explanation was, we admit, fanciful, and, as the event showed, faulty. In fact, it was impossible that it could have been correct, since the increased pressure during systole would have diminished the intensity of the murmur, even though it elevated the pitch. That the peculiar continuous murmur is pathognomonic of a varicose aneurism was recognized and maintained by Thurman. Mayne and Cossy equally urge its diagnostic value, though showing by their own cases that the symptom is not an essential one. In each the murmur was systolic only, which the writers explain on the ground that the very marked disease existing in the arterial walls interfered with the arterial contraction during the cardiac diastole, and in this way produced the absence of the diastolic part of the murmur. In fact, in several other

cases (VI., VII., IX, XV., XVII., XXVI., and probably II.), only a systolic murmur could be heard, and in still more (III., VIII., XI., XII., XIV., XVI., XIX., XXI., XXIII.) there was a distinctly double murmur, with an interval between the two parts. It is, therefore, evident that the continuous murmur is not an essential symptom of varicose aneurism, though such a valuable sign when present. The maximum intensity of the murmur, of whatever nature, was nearly always on the front of the chest, in the neighborhood of the first portion of the arch of the aorta, or over the abnormal centre of dulness. The only exception was the case of Christianu's (XXIII.), in which the maximum intensity of the double murmur was situated in the right supra-spinous fossa.

The method of the production of these different murmurs in the different cases is not at all clear. Sometimes they may have been due to the passage of blood through the perforation, audible only during a portion of the cycle; at other times they may have been produced in the aneurism or in diseased aortic valves. It is a noteworthy fact that in certain instances the signs of aneurism were very meagre. In the cases, namely, of Young's (IV.), Law's (V.), Wetterdal and Wallgren's (XX.), and Sisley's (XXVII.), there was neither thrill, dulness, nor murmur discovered, and in the last three no pulsation. Shannon's patient (XXII.) exhibited a diastolic murmur at the first examination, but after this no physical signs of aneurism could be found. In the case reported by Beevor (I.) no description of the physical examination of the chest is given, and that reported by Gulliver (XXIV.) was only a card pathological specimen, unaccompanied by clinical details.

Autopsy.—Little need be said under this heading, since the most important character—the perforation—was, of course, present without exception. The shape of the perforation in the different cases was slit-like, or more circular; its size never very great—half an inch or so in length or in diameter—and sometimes quite small. In some cases the edges were irregular and ragged, but oftener they were, to a certain degree, smooth, showing that in a comparatively short time after the rupture this smoothing can take place. In Glasgow's case (XIX.) the perforation was found closed by a thick plate of laminated fibrin. A fact worthy of note is that in nearly all the cases it is directly stated that compression of the cava existed as well as perforation. In Turner's case (XXI.) the cava was completely divided by the aneurism, so

that the distal portion looked like a short arterial trunk given off from the aneurism at its upper border. In our own case the production of this separation was well under way when the patient died. In Reid's case (III.) the perforation involved also the upper part of the right auricle, at the position of entrance of the superior cava. Another most important feature bearing on the production of murmur in aneurism in general is, that in only 5 cases (IV., XIV., XXI., XXV., XXVI) is disease of the aortic valves described.

Diagnosis.—Enough has been said about the characteristic symptoms. There is no condition with which the disease could be confounded except that of remarkably sudden obstruction of some other nature to the passage of blood in the vena cava, and such cases are almost hypothetical. From other varicose aneurisms of the aorta the diagnosis is easy. Rupture of an aneurism into the right ventricle or right auricle would produce general cyanosis and œdema. Rupture into the pulmonary artery could not produce the characteristic localization of œdema and cyanosis. The murmur would be heard at the left edge of the sternum, instead of at the right edge. The dyspnœa would probably be intense, and symptoms of engorgement of the lungs would eventually be followed by general anasarca, as in mitral disease.

Prognosis.—As the reported cases show, the prognosis is most unfavorable. Death has supervened in every case but one. In Shannon's patient (XXII.) only 6 hours elapsed before the fatal issue; in the case of Farrington's (XIV.), 13–15 hours; in that of Wetterdal and Wallgren's (XX.), less than 24 hours, and in that of Sisley's (XXVII.), less than 12 hours. On the other hand, patients may live days, weeks, or even months. Our patient lived 7 weeks; that of Kraus's (XXVI.), 40 days; that of Thurman's (II.), 2 months; that of Turner's (XXI.), 23 weeks; that of Glasgow's (XIX.), 7 months. In this last case the patient, after one month, seemed about to recover, then grew worse, and died. The patient of Damaschino and Lavin's (XVIII.) was living and practically well at the time the report was published. Although the murmur remained the same, collateral venous circulation had become established about one month after the onset of the symptoms, and œdema and cyanosis had about disappeared. The permanent recovery in this case, and the temporary improvement in Glasgow's case, indicate that the disease is not necessarily fatal, though the favorable chances are very slight.

Treatment.—This has proved useless. Venesection is the method which has been most frequently employed, but never with permanent benefit. Cupping, wet or dry, and free purgation may be employed. The strength should be sustained as far as possible, in the hope that the collateral circulation may become established, if the perforation be not too large, or that the opening may become closed by a thrombus.

ANTISEPSIS IN MIDWIFERY.

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On a former occasion I found from an examination of the records of the Health Department of New York City that the reported number of deaths from puerperal sepsis, between the years 1867 and 1875, in New York City, was 1947, or an annual average of 215.5. In the appended table, prepared for me from the same sources by my friend Dr. Rutsen Maury, it will be seen that for the five years from 1885 to 1889, inclusive, the deaths from puerperal sepsis reported were 1105, or an annual average of 221. The population of New York in 1870 was 942,292; in 1875, 1,169,305; in 1885, 1,553,730; in 1890, 1,755,292. It will be seen, therefore, that the ratio of deaths has by no means kept pace with the increase of the city's population.

A portion of this betterment is doubtless attributable to reforms in the management of our lying-in hospitals. Whereas formerly these institutions furnished nearly one-sixth of the fatal cases of puerperal sepsis, they now, when properly equipped and organized, afford the safest receptacles for parturient women. There has likewise been, without doubt, an improvement in results among the well-to-do classes. Among these there is a widely diffused belief that puerperal fever is a preventable disease, and that for its occurrence the physician should be held responsible, and this leads to greater painstaking on the part of the latter, even when he is disinclined to accept the deductions from modern scientific teaching. Among the poor, in their own homes, I do not believe that the dangers of childbed have been perceptibly lessened.

How serious is the question under discussion is shown by Dr. Maury's table, where it appears that, even in the past five years, one-tenth of the deaths in women between the ages of fifteen and forty-five are due to causes connected with childbirth, and that one-twentieth of

TABLE I.—DEATHS OCCURRING IN NEW YORK, BETWEEN THE YEARS 1867 AND 1875, INCLUSIVE, FROM METRIA, EXCLUDING OTHER CAUSES, WHICH PROVED FATAL DURING THE PUERPERAL PERIOD.

Year.	Jan.	Feb.	March.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Total deaths from metria.	Total from all causes.
1867	16	10	14	16	12	5	6	8	5	5	9	14	120	238
1868	17	14	27	16	18	9	12	8	7	3	8	12	151	267
1869	25	26	18	10	15	14	12	15	8	12	16	19	190	312
1870	12	17	37	27	20	10	12	16	10	12	11	17	201	333
1871	21	30	22	25	26	17	11	13	7	7	12	23	214	399
1872	26	37	41	40	27	21	14	24	13	14	18	32	307	503
1873	33	40	32	39	29	22	16	15	13	11	23	12	285	431
1874	19	32	24	33	34	21	18	15	10	7	7	25	245	439
1875	28	28	40	30	22	17	10	10	5	11	14	19	234	420
Total	197	234	255	236	203	136	111	124	78	82	118	173	1947	3342

TABLE II.—STATISTICS DERIVED FROM THE RECORDS OF THE NEW YORK BOARD OF HEALTH BETWEEN THE YEARS 1885 AND 1889 INCLUSIVE.

Year.	Total number of female deaths from any cause.	Total number of female deaths between 15 and 45 years of age—any cause.	Total number of reported births (including still-births).	Total number of deaths from puerperal causes, eclampsia, hemorrhage, sepsis, abortion, extra-uterine pregnancy, etc.	Total number of deaths from sepsis, puerperal fever, metritis, pyemia, etc.	Percentage of septic to puerperal deaths.	Ratio of deaths in pregnancy and childbed to entire number of female deaths.	Ratio of deaths in pregnancy and childbed to female deaths between 15 to 45 years.	Ratio of septic deaths to entire female deaths (all ages).	Ratio of septic deaths to female deaths, 15 to 45 years of age.	Ratio of puerperal deaths to reported births (with still-births).	Ratio of septic deaths to reported births.
1885	16,766	4,400	33,000	395	223	56½	1 in 42½	1 in 11⅛	1 in 75	1 in 19⅗	1 in 83½	1 in 152½
1886	17,082	4,307	34,369	384	199	51¾	1 " 44½	1 " 11¼	1 " 85¾	1 " 21	1 " 89½	1 " 173
1887	17,936	4,459	37,123	360	202	56	1 " 50	1 " 12⅓	1 " 88⅘	1 " 22	1 " 103	1 " 183½
1888	18,617	4,650	39,375	442	251	56¾	1 " 42	1 " 10½	1 " 74	1 " 18½	1 " 89	1 " 147¾
1889	18,390	4,644	41,867	430	230	53½	1 " 42¾	1 " 10 ⅘	1 " 80	1 " 20	1 " 97½	1 " 182
Total	88,791	22,460	185,734	2011	1105	55	1 in 44	1 in 11	1 in 80⅓	1 in 20	1 in 92¼	1 in 168

the deaths among women in the childbearing period are due to puerperal sepsis.

The following record will show how important have been the changes effected by modern methods in our lying-in institutions: In the year 1873 there were 449 births in Bellevue Hospital; there were twenty-five maternal deaths, fifteen of which were the result of septic infection. The proportion of one death from sepsis in thirty cases of childbirth was at that time the usual one in the maternities of this country and of Europe, reported differences being rather of book-keeping than of fact. There was always, likewise, a large unreckoned contingent of patients with pelvic inflammatory troubles which ended in recovery. In 1874, between the 1st of January and the 11th of June, of 166 patients confined, thirty-one died of puerperal fever. Similar fatal epidemics were at that time of not infrequent occurrence in the best appointed maternities.

The first attempted reform consisted in the erection of wooden pavilions on Blackwell's Island for the reception of the lying-in service. This move was the work of non-professional enthusiasts, and was the outcome of the prevailing opinion that a mysterious efficacy resided in wooden walls. The death-rate, however, speedily rose to a higher average than had previously existed at Bellevue; the wooden pavilions were abandoned, and the reign of inspiration ended.

With the transfer of the maternity to Blackwell's Island it became necessary to provide for the cases, sufficiently numerous, who seek hospital care after labor has actually begun. At first special arrangements for their reception were made with private institutions, but after a brief trial the latter refused, in justice to their other inmates, to admit such emergency cases, on the plea that it was impossible to receive them and keep their wards free from infectious diseases. Thereupon orders were issued by the commissioners that the patients should be taken on board the Blackwell's Island steamboat, there to be kept until they could be transferred upon the regular trips, which took place twice daily, to the Island maternity. Of necessity, many women were confined in the steamer cabin, especially during the night. No adequate provision was made for their comfort. Protests against the inhumanity of this arrangement received no attention, until, finally, in the winter of 1877–8, a patient died of cold, and became the subject of investigation by the grand jury. As a final outcome of

the agitation which followed, an old engine-house was fitted up for urgent cases, under the name of the Emergency Hospital. It contains a sitting-room, a laundry, and kitchen, a room for confinements, and a ward containing six beds. The visiting staff consists of Dr. Polk and myself. The attendance is derived from the internat on the medical side of the Bellevue Hospital.

I have ventured to offer these particulars because there is no pretence that the Emergency is a model for imitation. It has no fans for ventilation; no provision for a rotation of wards. It is by design intended for the homeless class. It receives, constantly, women with high temperatures due to infection before their entrance, or who have been days in labor, or who have been subjected to unavailing attempts at artificial delivery by outside physicians. It therefore furnishes an excellent test of the value of modern antiseptic methods. During the past five years there have been 837 women confined within its precincts, and there have been sixteen deaths, *i. e.*, nearly one to eighty-two. A glance at the record shows that these fatal cases are the best illustrations of the difficulties with which the institution has to contend, and of the value of the antiseptic principle.

Thus, there were five deaths from eclampsia. The patients were all sent to the hospital after the convulsions had gained full headway.

One case was a placenta prævia. The labor had been lengthy and the hæmorrhages profuse before the patient was admitted.

One was a case of Cæsarean section, performed on account of the obliteration of the cervix and vagina from cancer.

A patient was sent to the Emergency in labor, with delirium tremens. She died twenty-four hours after delivery.

There were two deaths following version in neglected shoulder presentations. Both women had been in labor several days previous to admission. The children were dead, and decomposition had set in.

In two cases the women had been respectively two and one-half and four days in labor when admitted. The children were dead. There was a stinking discharge, and commencing decomposition of the fœtus.

In two cases the high-forceps operation was performed surreptitiously by unqualified persons. In both instances the lower uterine segment was ruptured, and death speedily followed.

In a twin pregnancy, where one child was delivered by forceps, and

the second by version after a long labor, death occurred from septicæmia.

There remains one other case, concerning which no record has been preserved.

Thus, of the 16 deaths, 1 or possibly 2 can be construed as due to puerperal fever.

Now, the regulations by which these results have been obtained are comparatively simple. The patient, on her entrance into the hospital, receives a full bath. A rectal injection is given. The lower abdomen, the inner surfaces of the thighs, the pudenda, and the anus are scrubbed with soap and water, then washed with plain water, and finally cleansed with a 1 to 1000 solution of corrosive sublimate. The vagina is first douched with soap and water, and then with a 1 to 4000 solution of corrosive sublimate. In operative cases these douches are repeated before and after the introduction of the hand or instruments. The Bellevue Hospital internes, while on duty, are not permitted to visit the dead-house or the erysipelas pavilion. They are not allowed to assist at surgical operations nor to take part in the dressing of wounds. When in attendance upon a case of labor both interne and nurse wash the hands and forearms with soap and water, with pure water, and with corrosive sublimate solution (1 : 1000). Before an internal examination is made the hands are freshly washed with the bichloride solution. Infrequent examinations are recommended. Instruments are placed in a 2 per cent. solution of carbolic acid. After they have been used they are boiled and polished for future service.

The Credé method of expelling the placenta is employed. Ergot is given to secure good uterine contractions. In all cases after labor the vagina is douched with bichloride solution (1 : 5000). In cases of protracted labor, of high-forceps operation, of version, or of any manipulations by means of which air is admitted to the uterine cavity, the douche, after preliminary vaginal irrigation, is extended to the uterine cavity. The external parts are then dusted with iodoform, and are covered with a piece of gauze freshly wrung out in bichloride solution (1 : 5000). Outside is placed a pad of oakum. The dressing is changed once in six hours, at which time the genitals are scrupulously cleansed. During childbed no vaginal douches are given.

In the main the plan of treatment carried out is that introduced by

Garrigues at Charity Hospital five years ago. Its success is attested not only by the absence of deaths due to septic infection, but to that of morbidity in the wards. The temperatures rarely rise above 100½°, and, as a rule, do not reach 100°. But the plan of treatment presupposes intelligent instruments. For us these are supplied, on the one hand, by the training-school for nurses; on the other, by the hospital interne, who enters upon his duties thoroughly familiar with the requirements of surgical cleanliness. The occasional appearance on the scene of an individual who uses his "common sense"—*i. e.*, one who does not believe there is any harm in witnessing an autopsy, or in visiting a companion in the erysipelas ward, or in holding the pus basin in an operation for pyothorax—is almost certainly followed by temperature elevations, which add fresh testimony to the efficacy of a rigid observance of antiseptic regulations.

It is, however, to be acknowledged that there is by no means unanimity among authorities in relation to certain of the rules prescribed. Thus, the pad to the vulva is sneered at by many as savoring of the heresy that the air can become the source of contagion. For my own part, I believe an antiseptic dressing and pad is of immense service in hospital practice, where patients are aggregated together, as a means of preventing the decomposition of the lochia. The latter is inevitable upon the external parts when the ordinary napkin is employed. It seems to me incontrovertible that the air does thereby become contaminated, and it is doing violence to the evidence to deny the pernicious influence of a germ-laden atmosphere upon puerperal wounds. The argument that such an admission has a tendency to weaken the personal responsibility of the physician is childish. Aside from the fact that science is based upon truth, and not upon teleological considerations, the belief in a twofold origin of puerperal fever simply doubles his responsibility. To be sure, Leopold[1] found, of 427 patients in the Dresden Maternity who had not been examined or treated with corrosive sublimate previous to confinement, only 7 had a rise of temperature, and this fact taken by itself would seem to indicate that direct contact alone is capable of producing infection. But these patients were confined under favorable sanitary con-

[1] Leopold: Dritter Beiträg zur Verhütung der Kindbettfiebers. Arch. f. Gynäk, vol. xxxv. p. 149.

ditions. Szabo,[1] on the contrary, during an epidemic of fever in the Pesth Maternity, gave orders that no examinations should be made. Of 90 patients confined under these conditions more than one-third had febrile disturbances; and 2 of the patients, upon whom, in the one case, sutures, and in the other, serres-fines, were employed for perinea laceration, died. The researches of Ott[2] and Thomen[3] show that in healthy women the bacteria contained in lochia are found, not in the upper portion of the vagina, but near the introitus, which would certainly indicate that they are ordinarily derived from external sources. At any rate, since the use of the pad at the Emergency Hospital it has been possible to do away with the old-time rotation of wards, and the febrile temperatures, which once were so marked a feature of the winter months, when the windows were closed, are now no longer observable.

The subject of prophylactic douches during labor has likewise been one of endless contentions. Their employment at the Emergency has not, however, been the result of theory, but has been the outcome of the condition of the patients admitted, most of them having been examined outside by not over-fastidious midwives or physicians.

The differences which divide obstetrical authorities concerning the ante-partum douche are not so much the result of conflicting experiences as of doctrinal convictions. Thus, Barnes, Ahlfeld, Kaltenbach, and others, have shown that, in spite of every pains taken to exclude contact-infection, temperature elevations will occasionally occur in childbed. These febrile disturbances they regard as the product of a *materies morbi*, existent in the genital canal prior to labor; or, to use a term which at present is not only a designation but a war-cry, such cases are due to auto-infection. It is agreed to by all that in severe cases of puerperal infection the utero-vaginal canal swarms with bacterial forms, and that of these the streptococcus pyogenes is the most certain accompaniment of specific inflammatory troubles. When convalescence sets in, the coccus forms correspondingly disappear from the lochial discharges. But the vagina at all times contains a varied assortment of bacterial germs. Nothing would seem simpler, there-

[1] Szabo: Zur Frager der Selbstinfection. Arch. f. Gynäk, vol. xxxiv. p. 153.

[2] Ott: Zur Bacteriologie der Lochien. Arch. f. Gynäk., vol. xxxii.

[3] Thomen: Bacteriologische Untersuchungen Normaler Lochien, etc. Arch. f. Gynäk., vol. xxxvi. p. 247.

fore, than the deduction that these germs are always potential sources of evil. As a logical outcome of this doctrine its extreme supporters insist upon a complete sterilization of the vagina in every case of labor as an ordinary prophylactic precaution. Thus, Steffeck[1] advises the irrigation of the vagina during labor, at two-hour intervals, with a litre of corrosive sublimate solution. To increase its efficiency he introduces two fingers into the vagina to scrub, during the douche, first, the vaginal mucous membrane, and then the interior of the cervix. Döderlein[2] recommends that the vagina be first rubbed thoroughly with a preparation of creolin and mollin, and then be irrigated for ten minutes with a creolin solution.

Fortunately, these suggestions, which would have a tendency to handicap fatally the employment of antiseptic measures in midwifery, have not been received with much favor, even by the partisans of the doctrine of auto-infection. But, on the other hand, the pathogenic character of the germs contained in the vagina has been disputed. Thus, Artemieff[3] says that "the lochia of perfectly healthy puerperal women contains no microörganisms." Bökelmann[4] maintains that a healthy normal puerperal woman is *à priori* to be regarded as aseptic. He objects to attempted sterilization on the ground that the measures employed mechanically delay the progress of labor by the removal of the normal vaginal mucus, and at the same time render more vulnerable the tears, the excoriations, and the surfaces from which the epithelium has been detached.

Kaltenbach,[5] although a defender of auto-infection, states that in easy, rapid births the germs are carried away by blood, amniotic fluid, and the passage of the child; but in tardy labors, or in case of the premature rupture of the membranes, the entrance of germs into the uterine cavity is facilitated, and many may remain in the uterus and become a source of danger. Diseases resulting from auto-

[1] Steffeck: Ueber Disinfection der weiblichen Genital-canals. Zeitschr. für Geburtskunde, vol. xv. p. 395.

[2] Döderlein und Günther: Disinfection des Geburts-canals. Arch. für Gynäk., vol. xxiv. p. 111.

[3] Artemieff: Micro- und bacterioscopische Untersuchungen der Lochien. Zeitschr. f. Geburtskunde, vol. xvii. p. 174.

[4] Bökelmann: Die Antisepsis während der Geburt. Zeitschr. f. Geburtskunde, vol. xvii. p. 341.

[5] Kaltenbach: Arch. f. Gynäk., vol. xxxv. p. 489.

infection are lighter in character than those due to inoculation. Infection is first preceded by putrefaction. A complete destruction of germs is unnecessary; those that remain *in utero* are rendered harmless by ordinary injections. Even Winter states that in the present condition of our bacteriological investigations it is not right to carry out the attempts at complete sterilization recommended by Steffeck and Döderlein.

In private practice, under ordinary conditions, the presence in the vagina of infectious forms of bacteria is hardly conceivable, except in cases where they have been directly imported by the finger or by instruments. This statement is based both upon microscopic investigations and upon clinical experience. So far, it has not been possible, in the rare instances where beaded cocci have been found in the vaginal secretions, ante-partum, to obtain by pure cultures a product capable of giving rise to spreading inflammations; and in rural districts, even with not over-cleanly surroundings, immunity from infectious puerperal diseases is the rule. That, however, the vagina, which is not a hermetically closed tube, should, like the atmosphere, contain germs capable of producing putrefaction is hardly doubtful. Under healthy conditions the uterine cavity, on the contrary, contains no bacterial forms, and it is not invaded by them in normal parturition, a fact attributed to their feeble migratory power and to the downward current of the secretions. Conditions which favor the passage of germs from the vagina into the uterus, are relaxation of the uterine walls and membranes hanging from the cervix. The increased morbidity which accompanied the once popular vaginal douche in childbed was doubtless owing to the circumstance that, while of feeble disinfectant power, the injected stream furnished in many instances a direct highway to the uterine cavity. True midwifery antisepsis consists not so much in douching as in furthering physiological processes. Under normal circumstances a proper handling of the patient in the third stage of labor will effect more in the way of prophylaxis than Niagara. When, however, hands or instruments are introduced into the uterine cavity, the case is different. It would be hardihood then not to precede the operative measure by thorough vaginal disinfection.

In private practice too great pains cannot be taken with sanitary surroundings. The sheets and the napkin or pad to the vulva should be matters of personal supervision. Slovenly laundry work is danger-

ous. I prefer the pad because it is destroyed after having been once in use. The employment of napkins which have been left in a pail until putrefaction is well advanced, and are then soaked and dried before the fire by the monthly nurse, is a familiar procedure, and a direct temptation of Providence.

In city homes close inquiries should be made in reference to the condition of closets and sewer connections. My attention was first drawn to the similarity of typhoid and septic fever in puerperal cases by a patient sent to the hospital in a moribund condition. Her child had been removed by a high-forceps operation, and the cervix was badly lacerated. I regarded her symptoms as typical of puerperal fever, but the post-mortem examination showed a perfectly healthy condition of the pelvic organs and the characteristic intestinal lesions of typhoid. During the past few years of low water-supply in New York I have been many times called to see patients with high fever who were undergoing the customary douchings, while in the adjoining closet the basin, into which the dejecta of the patient were being thrown, was washed out once or twice daily by a pitcher of water poured into it by hand. The dangers of defective house sanitation have been ably presented by my friend Professor Playfair, and I find it difficult to understand the attitude of those who find no harm in the condition I have described because, they say, it does not affect the drinking-water.

A physician from the country called upon me to consult me about an epidemic of puerperal fever in his practice. I asked him many questions, but received no enlightening answer as to the cause. The physician was intelligent, and had seemingly taken every needful precaution. As he was about to leave me I inquired whether there was any diphtheria in his neighborhood. He answered, he had "lots of it," and seemed surprised that I should connect the diphtheria and puerperal fever together. It seems to me a thoroughly false idea that a physician can attend erysipelas, diphtheria, and scarlet fever, and at the same time pursue his midwifery practice with impunity, provided he makes free use of corrosive sublimate and carbolic acid. Indeed, I am almost disposed to regard as a special form of blindness the prevalent faith that parturient and puerperal women can only be infected by the finger and through the vagina.

The use of the vaginal douche in childbed as a routine practice is

now happily a thing of the past. Still, there is a widespread belief that intra-uterine irrigation is the true treatment for puerperal fever. But the case is not so simple. It is necessary to differentiate. We have just insisted on the fact that puerperal women are not exempt from febrile disorders other than those due to sepsis. Before the douche is used the breasts should be examined. The douche can help but little in acute and suppurative inflammations awakened by labor and in patients who have had chronic unilateral salpingitis antedating pregnancy. In cases of true puerperal infection, due to the penetrative form of micrococci and associated with spreading forms of inflammation, the use of the douche is probably idle and no more effective than washing the surface of the skin in an external erysipelatous process; and yet it is common to persist in the practice in spite of negative results.

The douche repeated at short intervals is not unattended with drawbacks. Thus, it interferes with the localizing processes by which most of the pelvic inflammations become self-limited; it helps to extend the morbid condition of the uterine cavity to the tubes, and, if continued long enough, paralyzes the uterine walls, so that after death the fundus is found to contain a dirty pool alive with bacteria.

The employment of the douche in fevers resulting from the absorption of toxine due to the putrefaction of clots, membranes, and bits of placenta within the uterus, on the other hand, is followed by immediate, favorable results and by the prompt disappearance of danger. But it is to be remembered that this is a clumsy attempt to remedy the results of past remissness.

The indications for the douche, as popularly employed, are especially vague. One man is governed by the odor of the lochia, but the odor in ordinary catarrhal endometritis is offensive, and yet all such cases recover, unless aggravated by the douche. Again, as Bumm has pointed out, the products of tissue-disintegration may be virulently poisonous and yet the odor may be absent. Others, again, use the douche when the temperature reaches 102°. But the temperature-guide leaves out of sight the multitude of temporary temperature elevations due to trivial local troubles, to psychical causes, and to malaria.

My own belief is that the intra-uterine douche in childbed is not indicated except when there has been slovenly help rendered by the

attendant at the time of childbirth. Usually a single douche is sufficient and should always be preceded by thorough vaginal disinfection. I still prefer the bichloride solution (1 : 3000), but inject slowly and not more than a quart. Then I take great pains to secure good contractions, in order to expel all of the injected fluid. I would strongly recommend, after the douche, the introduction of the iodoform "bacillus" (two and one-half drachms of iodoform). If the fever persists, it is sometimes desirable to follow the advice of Doléris, and explore the uterine cavity with the curette for retained bits of tissue. But it is time to stop treatment at this point. There is nothing more fatuous than the expectation of curing the patient by redoubling the vigor of the treatment.

DISCUSSION.

DR. A. JACOBI: It strikes me in Dr. Lusk's paper that the injury done by sepsis appears to be much less than I feel positive that it is. I know that I see between 40 and 50 cases annually myself in the easterly part of New York City. There are probably 1000 cases altogether in the eastern part of the city in the course of the year. I have seen in one week three in the practice of three different physicians. I took the trouble to find out how they got the disease, and I found they were attended by the same uncouth midwife. When the injury has been done then the doctor is called in. I believe Dr. Lusk would be doing a great and sacred thing if he would give some attention to the matter.

DR. WM. PEPPER: I can simply speak from the standpoint of the general consultant. I never attend cases of labor, but have, I am sorry to say, seen a great deal of puerperal fever in consultation, and I want to say a word of congratulation upon the emphatic condemnation of douching that Dr. Lusk has given. I am satisfied in my mind that the abandonment of that treatment has been followed by a distinct decrease of puerperal diseases. The important results reported by Dr. Lusk must be attributed to the avoidance of mischievous measures as well as to the adoption of excellent measures.

DR. J. WEST ROOSEVELT: As bichloride of mercury is poisonous in strong solutions and powerless to disinfect in weak, it is well to consider whether water is not safer and as efficient for irrigations. To employ a weak solution of a dangerous drug, which does not disinfect, seems irrational.

SEIZURES CHARACTERIZED BY SHOCK AND SUDDEN COMA.

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SHOCK may be defined as a condition of sudden depression of the vital functions, especially the cardiac, respiratory, and sensorial, by physical injury, or strong mental emotion. Coma is a state of unconsciousness, of profound insensibility, from which it is impossible, or difficult, to arouse the patient. The elements, then, of the class of seizures defined in our title are abruptness, violence, loss of consciousness, and depression of vital power. And these must be so developed and combined as to give character to the attack.

Syncope, with its characteristic feebleness of heart-action and cerebral anæmia, its constantly brief period of unconsciousness, its instantaneous and perfect recovery, its frequently trivial causation, and its trifling danger, is not usually reckoned a form of coma.

Coma, with more or less of shock, is observed as an incident of many diseases which it cannot be said to characterize. It may occur during the invasion, in the course of, or near the termination of a disease. In the last case it may be simply a manifestation of the final failure of vitality. It occurs in a great variety of diseased cerebral conditions, especially concussion and compression, anæmia and hyperæmia, extravasation of blood and serous effusion, inflammation, exhaustion, and sunstroke. It is found related to various convulsive affections, as epilepsy, hysteria, infantile reflex, puerperal and uræmic convulsions. It is common in connection with fevers, both essential and symptomatic, continued and malarial, especially when hyperpyrexia is present. It is seen at the invasion of scarlatina and other eruptive fevers, often associated with convulsions in the case of children.

Uræmic coma is usually preceded by or accompanied with convulsions, and generally terminates fatally. Diabetic coma is sometimes met with, usually toward the end of the disease, and is often preceded by marked nervous phenomena. It usually progressively deepens to a fatal issue within two or three days. The breath is apt to take on a characteristic fruity odor, sometimes suggestive of chloroform or ether. It is, doubtless, due to the formation in the blood of a peculiar poison, believed by some to be acetone. Uremia may occur as a complication in diabetes, but does not account for the diabetic coma.

Cholæmic coma is chiefly seen in the so-called "grave jaundice," and is believed to depend upon the accumulation in the blood of the excrementitious element cholesterin, a product of the disassimilation of nerve tissue. Death, with or without convulsions, is the usual result.

Anæmic coma is oftenest seen in pernicious anæmia. In its clinical features it closely resembles diabetic coma. It is believed by some also to depend upon a condition of acetonæmia. Owing to deficiency of red corpuscles the process of tissue oxidation is imperfectly carried on, and the production of toxæmic substances is thereby rendered probable.

Coma dyspepticum is a rare affection, believed to result from poisonous gases, acids, or ptomaines, formed in the gastro-intestinal tract. The coma is usually preceded by dyspeptic phenomena, muscular weakness, headache, and joint-aches. The peculiar fruity odor of breath and urine, common in diabetic coma, is present. Gradual recovery often occurs, after from two to four days of unconsciousness. Closely related to this form of coma is that associated with the presence of septic, decomposing pus in natural or artificial cavities of the body.

Mention has been made of the presence of coma in a variety of diseased conditions, believed to result from the presence in the blood of poisons of autogenetic origin. It may be sufficient now simply to suggest the frequent occurrence of coma as the result of poisons introduced into the system from without, most notably alcohol, opium, chloral, and cannabis indica.

The main object of this paper is to collate and compare, chiefly as to clinical history, the four typical forms of apoplectiform seizure, viz.: the hemorrhagic, the embolic, the congestive, and the uræmic.

I. The Hemorrhagic Form.

In the discussion of this variety of apoplectiform seizure I shall not include the minute diagnosis of hemorrhage in the rarer possible localities of the brain, but shall, for present purposes, assume it to be in its usual site, in and about the basal ganglia, involving the nucleus caudatus, the internal capsule, the nucleus lenticularis, the optic thalamus, and the immediately adjacent parts. A probable explanation is that these parts receive their blood-supply through large and directly penetrating branches of the anterior, middle, and posterior cerebral arteries, with high intra-vascular tension. The walls of the small vessels are often the seat of atheroma and endarteritis deformans. Miliary aneurisms, of size from that of a milletseed up to that of a pinhead, form and burst. The hemorrhage occurs in two forms, the capillary, with its separate fine red dots, and the focal, or clot form, the clot being sometimes no larger than a pea, and sometimes occupying the greater part of a hemisphere. The lateral ventricle is often opened into. The traumatic injury inflicted upon the brain tissue; the anaemia induced by clot pressure; the clot-changes, resulting finally in a mere pigmented cicatrix, with or without the intervention of a cyst; and the later secondary lesions of descending degeneration, involving the crossed, and, in less degree, the direct pyramidal columns of the cord, must not be forgotten.

In connection with the question of causation let us keep in mind that so-constantly-present, double, ultimate cause, degenerated vascular walls and intra-vascular pressure; the possibility of an existing hemorrhagic diathesis; the age and sex most obnoxious to cerebral hemorrhage; the influence of alcohol, syphilis, gout and heredity; and the tendency of muscular and emotional strain.

The events of the clinical history of cerebral extravasation occur to the mind in a certain natural order of sequence. Thus we have (1) the shock, (2) the period of unconsciousness, (3) the condition of the patient as he emerges from the coma, (4) the special liabilities of the first week, (5) the remaining-over symptoms, and the gradual, usually incomplete recovery, (6) the later phenomena of descending degeneration of the pyramidal columns, and (7) the special liabilities of the patient in subsequent life.

The popular term, "shock of paralysis," is an entirely natural and

appropriate one. The sudden depression of the vital functions, which the word shock implies, is coincident with the vascular rupture, and the amount of shock must bear some proportion to the size of the artery ruptured, and the amount of injury inflicted upon the brain tissue. Sometimes the primary shock is comparatively slight. The patient may feel confused, or even fall, but quickly recovers himself in a measure, and is able to talk intelligently till advancing coma finally overcomes him. The elements of shock and coma are so related and combined in these cases that it is impossible to draw a sharp line of separation. The apoplectic invasion is sometimes absolutely abrupt. A man feeling at his best, engaged in brilliant conversation, or dexterously performing some delicate operation, may fall to the floor unconscious about as suddenly as if he had received a heavy blow upon the outside of his head. In another case the coma may be of gradual and progressive development, an hour being occupied in the process.

In such a case the blood escapes slowly from the ruptured vessel, or there may be several successive bleedings of only moderate amount. The coma is largely due to the suddenness of the pressure exercised by the extravasated blood. It may, therefore, be more profound and protracted in proportion as the effusion of blood is larger and more rapid. Sometimes the seizure is preceded for days, or hours, by disordered sensations of the head, such as headache, dizziness, a sense of fulness, or flushing of the face. Occasionally an attack occurs during sleep, and the patient is found unconscious and hemiplegic.

The face is usually red or livid, but exceptionally it is pallid. The breathing is generally slow and stertorous, and its rhythm is broken and irregular. If the coma be profound the lips and cheeks are flaccid, and the latter are puffed out in expiration. The condition of the pupils varies. Most often, perhaps, they are dilated; but they may be contracted, or of natural size. Some disparity between them is not unusual. The face is often twisted, the angle of the mouth being drawn toward the non-paralyzed side. The pulse is not constant in character, but is often full and hard. Vomiting is common at or soon after the seizure. The body heat, which is often reduced 1° to 2° at first, usually reaches several degrees of elevation within twenty-four hours. Even in the stage of coma the existence of hemiplegia can frequently be recognized. Slight unconscious movements of the limbs on one side occasionally occur, while those of the opposite side are

absolutely motionless. The muscles of the paralyzed side are usually limp and flaccid, but are rarely in a state of tonic rigidity, the latter condition being specially related to the bursting of effused blood into the lateral ventricle. Convulsive movements are rare unless the motor cortical region is involved. Conjugate deviation of the head and eyes toward the side of the hemisphere involved is occasionally present, especially when the severer lesions exist.

The coma is usually recovered from within a few hours. Exceptionally, death occurs in from a few hours to a day or two, the coma persisting to the end. In these cases the cardiac and respiratory centres of the medulla are presumably involved in the hemorrhage. As a rule, if no improvement in the coma occur within eight or ten hours, it grows more profound up to a fatal issue. Death occurs by apnœa, or by blended apnœa and asthenia.

The mental condition of the patient emerging from the stage of coma is dull and apathetic. He asks no questions. He expresses no surprise. He seems to be utterly devoid of recollection or conception pertaining to the experience he has just passed through. In this respect the condition is in marked contrast with that of one emerging from the influence of ether.

The special liability of the first week is to the occurrence of inflammation of the brain-tissue around the clot. The cerebritis manifests itself by fever, headache, delirium, and spasmodic contractions of the paralyzed muscles. In the worst cases convulsions may occur, and death by coma, but in the great majority of cases the inflammation subsides in the course of a few days.

With the full recovery of consciousness comes the opportunity to the physician to take competent note of the remaining-over symptoms, and to "estimate the damages." The most common of the persistent symptoms of hemorrhagic apoplexy are hemiplegia, aphasia, and impairment of mental power. So far as these are permanent, they are the result of the primary traumatic injury inflicted upon the brain tissue at the moment of extravasation, which is finally represented by the apoplectic scar, or cyst. These are the direct focal symptoms. Then there are indirect focal symptoms, resulting from temporary conditions, such as clot pressure, œdema, etc. These indirect symptoms disappear in the course of days, weeks, or a few months at the farthest.

The hemiplegia is the most important and constant of the persistent symptoms. It is present in a large majority of the cases. The muscles ordinarily involved are the muscles of the upper and lower extremities, and of the nose, cheek, and mouth. The trapezius muscle is often paretic, inducing sagging of the shoulder. The respiratory muscles of one side may also be paretic, and marked disparity in the amount of respiratory play on the two sides may be noticed, especially in forced breathing. These paralyses are all on the side opposite that of the hemorrhage. The motor function is solely, or chiefly, affected. That of sensation is usually little, if at all, involved. There is great variety in the degrees of completeness of the paralysis. It ranges between absolute loss of motor power and mild paresis. The upper limb is, as a rule, much more seriously affected than the lower. There is wasting of muscle even in the early weeks. It is the direct result of the brain lesion and is in some proportion to the degree of paralysis. It is an atrophy of disuse. It is in marked contrast with the extreme wasting, with associated degeneration of tissue, of later occurrence. Reflex movements occur, especially in the lower limb, even when the motor paralysis is complete. The tendon-reflexes are exaggerated, while the skin-reflexes are reduced. Electro-muscular contractility is maintained. So, also, is the direct mechanical excitability of the paralyzed muscles. The pulse is observed to be smaller on the affected side. The temperature is reduced. The natural color of the skin may be replaced with a purplish hue. Distention of the veins and œdema may be present. The nail-growth, as pointed out by Weir Mitchell, may become arrested. Temporary irritative contractures may occur. There is a tendency to bedsores, especially over the trochanter and the nates of the paralyzed side, in severe cases.

The facial paralysis is usually on the same side as that of the limbs. It affects only the parts supplied by the lower division of the facial nerve, the nose, cheeks, and mouth. The expression of the face is lost. The twist is sometimes very pronounced. In other cases it is only noticeable when the patient laughs or cries. The tongue, when protruded, points to the disabled side, the genio-hyo-glossus muscle on that side being paralyzed. The muscles of the palate may be affected. Ptosis and strabismus are occasionally present on the side opposite that of the paralyzed limbs.

Aphasia is sometimes the prominent symptom. The power of

speech may be absolutely gone, or, it may be greatly modified and restricted. The memory of words may be partially or completely lost from the mind (amnesic aphasia), or the power of articulate speech only may be wanting (ataxia aphasia). There may be a stumbling over syllables, so far, for instance, to call a widow a window (literal ataxia) The mental association of individual ideas with particular words may be broken up, leading to the indiscriminate use of words (paraphasia). There may be word-deafness, so that the vernacular may sound to a person like a foreign, and unknown language; or word-dumbness, the person being unable himself to call up words, while quite competent to comprehend words spoken by others. There may be the coining of a single meaningless phrase, which is made use of on all occasions, and for all purposes of expression (monophasia). Modifications of speech-power appear in every conceivable form, and with every possible limitation. The allied disturbances of agraphia, amimia, and alexia are often associated with aphasia. Abstractly speaking, aphasia and mental imbecility are entirely distinct affections, but I think the former can hardly exist, in any considerable degree of development, without some association of the latter.

An interesting case of monophasia occurred in my practice some fifteen years ago. The patient was a man not far from forty-five years of age. A week before he had experienced an apoplectic shock. He had right-sided hemiplegia and aphasia. He had been lifted up into an arm-chair. His vocabulary consisted of three words, "yes," "no," and "boudeegy." His wife proposed that he should read me his favorite hymn, and he read with great animation and satisfaction four verses, as follows:

Boudeegy, boudeegy, boudeegy, boudee,
Boudeegy, boudeegy, boudee,
Boudeegy, boudeegy, boudeegy, boudee,
Boudeegy, boudeegy, boudee.

He looked up at me eagerly for sympathy in the sentiment. I saw him repeatedly within the next few months. The hemiplegia passed off. His health was restored. The aphasia remained. Ten years later I met him in the cars. He greeted me with a warm shake of the hand, a bright expression of the face, and the single word "boudeegy." He introduced me to the gentleman sitting beside him, "Boudeegy—Boudeegy." "Glad to know you, Mr. Boudeegy," said

I, and we shook hands cordially. He had opened a village store, and was engaged in successful business. He controlled affairs. His clerk did most of the talking. He had added, as I learned, one word to his vocabulary. Under extreme provocation he would say "damn." Last summer I met his old physician, who told me there had been a religious revival in the village, our patient had become interested, and had left off saying "damn." He had reduced his scanty vocabulary twenty-five per cent. for conscience' sake.

Aphasia is sometimes, though rarely, present without hemiplegia or coma. It may constitute the only obvious manifestation of a slight cerebral lesion. I saw an instance of this sort a few years ago. The patient was a lady about seventy years old. She had suddenly lost the power of speech. There were no associated phenomena. Her intelligence was perfect. Her natural manner, which was peculiarly sprightly and attractive, had lost nothing of its individual charm. She was speechless. That was all. The aphasia lasted some six weeks, when one morning she astonished her son-in-law, who had entered her chamber, by remarking, "Charles, that's a match-box on the mantel. I've been trying to get hold of that word for a month." The recovery was as complete as it was sudden. The cerebral lesion in this case must have been slight. Was it hemorrhagic, embolic, or thrombotic which?

Impairment of mental power is a frequent permanent result of hemorrhagic apoplexy. Even in instances spoken of as perfect recoveries, it may well be doubted whether the full, original power of achievement and endurance is regained. There generally results a weakening of the will-power, while the emotions are excitable and less easily controlled, and the temper is irritable and moody.

In the lighter cases of cerebral hemorrhage improvement soon begins. The indirect focal symptoms promptly disappear, and those directly resulting from the primary brain injury may gradually diminish, until, in the course of two or three months, the patient may consider himself as perfectly recovered. The sensibility returns first, and often very quickly. The facial paralysis may rapidly diminish or disappear. Improvement is earlier and more complete in the lower than in the upper limb.

In the severer cases, when a fatal result does not occur within the first week, the paralysis, aphasia, and mind-weakening may become

permanent. The hemiplegia is likely to be more considerable and permanent in proportion as the primary injury to the internal capsule is more extensive. The hemiplegic gait is peculiar. The body is inclined upon the strong limb, while the paralyzed limb is dragged after it with a tremulous jerk and swing, the depressed toe causing a continual liability to trip.

Later in the history of cerebral hemorrhage attended with persistent paralysis, permanent contractures of muscles occur. These affect the upper more than the lower limbs. Associated with them are marked atrophy and tissue-degeneration. Some authorities, notably Charcot, attribute these changes to a descending degeneration of the pyramidal columns of the cord, while others regard them as "passive contractures," resulting, like the permanent paralysis itself, from the primary injury of the pyramidal tract. As pointed out by Weir Mitchell, "post-hemiplegic chorea" often involves the paralyzed muscles.

Persons who have had one attack of cerebral extravasation are very likely to have repeated ones. The degenerative process in the vessel-walls is progressive. Each seizure is more likely than the preceding one to end fatally, and each one, if not directly fatal, entails new degradation of vital power. Death by coma comes about in this way, or some intercurrent disease carries off the patient.

II. Embolic Form.

The middle cerebral artery—oftener the left—is the usual site of brain embolism; rarely the internal carotid, or the basilar artery. Embolic softening, when it occurs, involves, in the great majority of cases, the same parts usually involved in cerebral hemorrhage, including the internal capsule and the great central ganglia. The first direct effect is anæmia of the brain-tissue usually supplied by the plugged artery. If no relief be afforded by the establishment of collateral circulation within twenty-four hours, the process of necrobiosis commences. Fatty degeneration and softening occur and the tissue perishes. If the injury inflicted upon the brain be not too extensive, and the patient live, the dead matter is removed by absorption, and may be replaced by a cyst or cicatrix, as often seen after cerebral hemorrhage.

If the emboli be infectious or gangrenous, gangrene or abscess may be expected to follow. The chief sources of the emboli are endocarditic

and thrombotic deposits upon the mitral and aortic valves, atheroma of the walls of the aorta and large arteries at the base of the brain, and aneurism of the aorta.

The clinical history of the embolic form of apoplexy bears a very close resemblance to that of the hemorrhagic form. Indeed, in some cases a positive diagnosis between them is impossible.

The primary shock is the same in kind in both cases. The onset is sudden and absolutely without warning. There are none of the cerebral premonitions so often present in hemorrhage of the brain. When vascular and nutritive lesions exist in the brain, it is only a matter of coincidence. The embolus is of other and distant origin.

The intensity of the shock varies greatly in different cases. There may be only slight confusion of mind, or there may be profound coma. In perhaps half the cases coma is absent. The unconsciousness, the stertor, the slow pulse, the vomiting, and the immobility and irregularity of pupils may all be present, but they are not so frequently present, nor, as a rule, so pronounced as in hemorrhagic apoplexy. Exceptionally the primary shock is slight, and the coma is more gradually developed, as if the embolus were small and required reinforcement, by the formation of a thrombus about it, before becoming competent to produce marked effects. The coma is not usually so long continued as in extravasation, and instances in which it remains till a fatal issue are very rare.

The symptoms remaining over, after the passing off of the coma, are the same in the embolic as in the hemorrhagic form, viz.: hemiplegia, aphasia, and impairment of mental power.

The hemiplegia is the most constant symptom. The paralysis is usually on the right side of the body, always on the side opposite that of the brain lesion. The upper limb is usually more affected than the lower. Involvement of a single limb only is less frequent. The face may or may not be involved. The paralysis may be permanent, as is so often the case in extravasation, but more frequently it is quickly and sometimes completely recovered from. Great individuality is given to cases by varying combinations of the four essential elements of shock, coma, paralysis, and aphasia.

Aphasia is present, in association with hemiplegia, with great frequency. It may occur in any of its forms and in any degree of development. It may also occur as an almost solitary symptom with-

out the association of either coma or hemiplegia. In such cases prompt and complete recovery often ensues. The anatomical basis may be some slight thrombotic rather than embolic lesion. Epileptiform convulsions are said to exist more frequently, as a complication, in embolism than in extravasation.

It is doubtful whether organic aphasia, any more than extravasation, ever exists without some resulting permanent loss of mental and physical power. The degree of such loss varies with the extent and location of the cerebral lesion. It is never so great from embolism as from extensive hemorrhage.

When recovery has been made from a first attack of cerebral embolism, resulting from chronic cardiac or vascular disease, the permanent liability of a repetition must remain.

In making the differential diagnosis between embolic and hemorrhagic apoplexy, the following-named circumstances would have weight as pointing toward embolism, viz.: (1) absence of cerebral, prodromic symptoms, such as vertigo, headaches, face-flushings, etc.; (2) history of preëxisting cardio-vascular disease; (3) coëxisting embolisms of other organs, as retina, or kidney; (4) the occurrence of hemiplegia alone, without sudden loss of consciousness; (5) lightness of shock; (6) rapidity and completeness of recovery; (7) early life.

III. The Congestive Form.

The pathological basis, in this form of seizure, is distention of the cerebral vessels with arterial blood. The actual state of the affected brain-tissue is that of anæmia, from pressure of the distended arteries. Passive congestion of the brain, in which the veins and sinuses are crowded with venous blood, as the result of cardiac, or other form of obstruction, is not a direct primary cause of apoplexy. It produces it sometimes indirectly by favoring the occurrence of hemorrhage.

Circumstances predisposing to arterial cerebral congestion are general plethora, a sanguine temperament, a short, thick neck, and the age of early maturity. The most common exciting causes are excessive muscular, mental, and emotional activity; pure, non-compensatory hypertrophy of the left ventricle of the heart; over-feeding, with lack of exercise; alcoholic over-stimulation; and vaso-motor influence. Obstruction to the natural flow of blood in the arteries of the organs and parts may also lead to excessive flow into the arteries of the brain.

Prodromic symptoms are quite constant—such as headaches, a sense of cerebral fulness and throbbing, noticeable heating of the carotid and temporal arteries, flushing of the face and eyes, heavy heart-action, asomnia, mental confusion, nervousness, and irritability.

The seizure is marked by shock and unconsciousness, as abrupt and often as pronounced as in extensive hemorrhage into the brain. The face is flushed, the eyes are injected, the pupils are contracted, the pulse is full and bounding, the breathing is heavy and stertorous, and the throbbing of the carotid is visible, as well as palpable. There are no unilateral phenomena.

If prompt and judicious administration be made of sedative and depletive treatment, relief is usually quickly obtained. The liability of vascular rupture and extravasation constitutes the chief danger. The recovery may be complete, but the necessity of careful regulation of the diet and regimen, with a view to prophylaxis, is imperative.

The congestive form of apoplexy is distinguished from the hemorrhagic and embolic forms by the distinctive group of symptoms connected with the seizure, and by the absence of the remaining-over symptoms, hemiplegia, aphasia, and impairment of mind. It is separated from the uræmic form by the absence of albumin and tube-casts from the urine, and its freedom, as a rule, from the convulsions so common in uræmia. The differential diagnosis from alcoholic coma would be helped by the exclusion, both from history and environment, of everything suggestive of alcohol.

IV. The Uræmic Form.

The anatomical basis in this affection is the contracted kidney, or the kidney of diffuse chronic nephritis.

The essential pathological condition is toxæmic. There is an accumulation in the blood, and also probably in the tissues, of urea, or at least of some one or more of the excrementitious products of tissue-metabolism, of which urea represents the final stage, which are normally thrown off in the urine.

The coma of uræmia is sometimes gradually developed without sudden attack. In other cases coma occurs with apoplectic suddenness. It is only with the latter class of cases that we are now concerned.

There may be prodromic phenomena—such as headache, dizziness,

dyspnœa, dyspepsia, œdema, or a fleeting sensation as if about to faint. But the seizure is abrupt, with marked shock. A man of early or middle life, of pale complexion, and contrasting in physique with a subject of congestive apoplexy, who has never thought of himself, or been regarded by his friends as a sick man, is suddenly stricken down. Later—in retrospect—friends may be able to recall little circumstances indicative of slight impairment of vigor and elasticity of mind or body, which did not at the time arrest their attention. The coma is profound. The face is pale and often covered with cold sweat. There is stertorous breathing, and the pupils are dilated.

In the great majority of cases the coma is preceded by, or accompanied with convulsions. These are epileptiform in character, with frothing at the mouth and biting of the tongue. The convulsions may be repeated at longer or shorter intervals, and death may occur within two or three days, with no recovery of consciousness. Or, the coma being less profound, consciousness may be recovered, and weeks or months may elapse before the occurrence of the final and fatal seizure. During this interval the characteristic anæmia of chronic Bright's disease may become rapidly developed.

Hemiplegia and other symptoms, which characterize cerebral extravasation and embolism, are usually absent. So also are the peculiar physique, the flushed face, the injected eyes, the contracted pupils, the full, bounding pulse, and the other characteristic symptoms of active cerebral congestion. The finding of albumin and tube-casts in the urine, which should be early drawn by the catheter and tested, completes and establishes the diagnosis.

DISCUSSION.

Dr. H. M. Lyman: I remark that the cases of apoplectiform seizure, to me the most interesting because most difficult of explanation, are those which have been but little touched on in the paper.

I saw, not long ago, an elderly gentleman who awoke in the morning in the possession of all his faculties, not paralyzed but complaining of numbness occupying the whole left side of the body. There was reduction of sensibility, which was not completely destroyed but greatly impaired. His mind was clear. There was very little difference between the two halves of the body so far as the muscular movements were concerned. I was inclined to think there was

no rupture of any vessel, but that there had been some change, some obstruction, perhaps, of the circulation. I could not localize it.

The reference to aphasia reminds me of a case coming under my notice. He was a man about sixty years of age, active, energetic. One day he had occasion to trundle his buggy into the barn. The hired man did not do it to his satisfaction. He jumped forward, seized the axle, and pushed it up an inclined plane into its place. He said he immediately felt a pain in his head. He could walk as well as ever, and could use his arms; there was no paralysis excepting a form of aphasia, in which he knew what he wanted to say, but many of the words that he desired to use escaped his recollection. He finally recovered.

Dr. A. L. Loomis: Dr. Dana has brought before us a very comprehensive subject. It seems to me that the diagnosis between cerebral apoplexy, cerebral embolism, and cerebral thrombosis is often a very difficult one. I found, a few years since, in a series of cases in which hemiplegia was marked and in which coma was present at the onset of the attack, the pathological lesion of thrombosis and softening where I had expected to find apoplexy. I have also been astonished to find that cerebral apoplexy after middle life is not of so frequent occurrence as I had supposed. The majority of cases of so-called apoplexy in the aged I believe will be found to be cases of cerebral thrombosis and softening.

A sudden hemiplegia coming on before fifty years of age is undoubtedly, in a majority of cases, if not all, due to extravasation. Coma coming on suddenly with hemiplegia and facial paralysis late in life is more likely due to thrombosis. So far as uræmic coma is concerned, there are so many different conditions of the brain when it occurs that I hardly think we can say that it is always due to toxæmia. Congestive apoplexy is a condition which I can hardly understand.

Dr. R. T. Edes: Within a few years there have been a number of cases reported in connection with uræmia, in which symptoms pointed to a localized lesion, but in which none of the usual conditions, such as hemorrhage or thrombosis, have been found. For these a localized cerebral œdema has been suggested as an explanation.

Œdema of the brain after death might disappear very easily, and consequently the not finding of it would not be conclusive against this hypothesis. The rapid occurrence and subsidence of local external œdemas in patients of this kind would seem to point toward the possibility of such a condition elsewhere.

Another point I desire to comment on is the mental condition in cases of aphasia, and the possibility of definite thought without words. Lordat, who himself was affected with aphasia, insisted that though he had no words on his tongue, he was able to compose carefully a lecture in all its parts without a single word going through his mind; but Trousseau, in commenting on this case, thinks that the learned professor may have overestimated the accuracy of his mental processes.

I heard a paper read a short time ago in which the reader took the ground that in every case of aphasia there is more or less mental impairment. I am inclined to think this is not always true, The mental operations may be found. I think there is more of a reluctance to the exertion of thinking than inability to guide the thoughts clearly and logically. A gentleman of prominence in our own profession, after an apoplectiform attack, although he has not complete aphasia, is almost sure to lose the essential words in a long sentence. Yet his mind is clear. His advice and assistance are sought in various kinds of business. He seems just as much annoyed as anyone else would be by the failure to get the word. Macready (*New York Journal of Medicine*, September, 1857) gives a number of cases of this kind.

DR. A. JACOBI: Do the cases just mentioned occur in infancy, childhood, or old age?

DR. EDES: Those I refer to are in adults.

DR. JACOBI: All the cases I have seen were of adults. I have seen three or four cases like those described by Dr. Edes.

DR. JAMES TYSON: I appreciate the feeling that with such positive symptoms as exist in uræmic coma and convulsions there ought to be some anatomical change associated. But while it is true that the theory of pressure from acute œdema may explain such symptoms, the demonstration of its presence after death is difficult or impossible.

On the other hand, we have only to recall the phenomena of chloroform or ether intoxication for a picture identical with what is often seen in uræmic coma. Yet I do not think anyone would claim that there is any anatomical alteration responsible for them. We are forced, therefore, with our present knowledge, to conclude that there is a toxic something in or out of the blood-vessels which, by its action on the nerve-cells of the brain, is responsible for such symptoms.

DR. I. T. DANA: I do not want to take up any more time. I will say, while the name of the paper opens up a broad field, yet it is limited by the title to those cases that were accompanied by shock. I will only express my thanks to the friends who added so much interest to the paper by their discussion of it.

SOME DISORDERS OF SLEEP.

By S. WEIR MITCHELL, M.D., LL.D. Harv.,
of Philadelphia.

When I too lightly undertook the task of referee in the debate on Sleep and its Disorders, I did not realize how large was the labor involved; nor, indeed, how difficult. When dealing with organs or functions the physiology of which we, in a measure at least, comprehend, it becomes easy and pleasant to discuss their alterations from health; but as regards sleep we know little. The wildest theories have been entertained concerning it; and, after all, we are simply driven to believe that it is a state of the nerve-cells—and why not of the nerves?—in which they become functionally actionless in a variable degree. Whether this be true also of the other cell-structures of the body we do not know: and sleep may be a universal function, as would seem reasonable to those who believe that plants sleep. It is sure, also, that the sleeping brain contains less blood, or it circulates less, than the brain awake, and this is the limit of what we know.

The disorders of sleep are many. I have found myself driven to choose among them, and not more than will occupy my share of your time. Leaving to my co-referee abnormal sleep—the too much and the too little, dreams, nightmares, and somnambulism, I shall limit myself to a less known group to some members of which I was the first to call attention.

In 1876[1] I described several of the morbid states of sleep, and again wrote of them more freely in 1878.[2] In 1882, in my lectures on "Nervous Disorders of Women," I dwelt at greater length on the symptoms in question, as well as on others which have been much discussed of late in the journals without notable additions of value.

In making choice of how I shall treat of sleep troubles, I have

[1] Phila. Medical Reporter, 1876.

[2] Virginia Medical Journal, 1878.

been influenced somewhat by the fact that certain of these it has been my fortune to see and to study more than I have the better known phenomena of dreams, somnambulism and the like. Moreover, Dr. Folsom will, I believe, deal with much that I do not handle, and, between us, we shall, I trust, reasonably well illustrate the disorders of sleep.

The approach to the unconsciousness of slumber, and, too, the return from it to the world of volition, may be medically considered as part of sleep, and, as I shall show, these periods are often disturbed by certain very interesting symptoms.

As we are falling asleep the senses fall from guard in orderly and well-known succession—this interval I desire to label the *præ-dormitium.* When we begin to awaken, and the drowsied sentinels again resume their posts, there is a changeful time, during which the mind gradually regains possession of its powers—this interval I may call, in like fashion, the *post-dormitium.*

The Relation of the Præ-dormitium to Insanity.

In the borderland of coming slumber, when we are not yet overwhelmed by its full power, the steadying contradictions of the external world are, in a measure, by degrees cut off, whilst the will still holds a slowly lessening rule. It has long been known to alienists that the præ-dormitium is apt to be invaded by hallucinations in those who are about to become disturbed in mind. Every student of himself knows, too, what a fairy country for visions is this intermediate state. Since, in the well, it is the time for castle-building, it seems natural that, in the disordered, it should serve to foster dangerous hallucinations, and that, in rare instances, these should be limited to the period in question. Baillarger is the only author who has studied with any care the relation of the præ-dormitium to insanity. Of this he says:

"The organs of sense ceasing to transmit to us exterior impressions, the control of our ideas escapes us, and whatever rises appears, as it were, spontaneous; at times vague or confused fantastic forms succeed one another, and we have of it all but a half consciousness. At times more distinct forms appear, and we are present, as it were, at a strange spectacle in which we take no active share, but which leaves distinct traces on the mind. Any exterior intervening impression causes these visions to vanish. A sudden

noise, or touch, or light awakens fully the senses, recalls attention, and these phantoms are effaced."[1]

In a long series of interesting cases he shows that certain persons otherwise still sound, are liable to have between waking and sleep hallucinations which long precede the outbreak of insanity. He describes instances of such hallucinations of sight and hearing as lasting from one to three years, and ending in grave mental disease. These were usually voices or visions fading when the eyes were opened. In one case there was something like the sense of a blow on the head, and then on the bed, but nothing comparable in this direction to the phenomena of sensory shocks to which I shall presently call your attention.

I have myself seen illustrations of the facts mentioned, and I speak of them here because this is ground we rarely go over in our examinations of patients. It will possibly be found that valuable prognostic indications as to insanity may be gained by examination of the præ-dormitium.

Before passing on to other matters, I may say that generally, as I have known them, these prodromes of insanity were connected with eye or ear alone. In but one case was olfaction concerned. I will content myself with a sketch of it.

CASE I.—Mrs. C., æt. forty, of a neurotic family, all liable to neuralgic headaches. One brother died of ataxia. Convulsions in infancy were common to all five brothers and sisters—none ending in epilepsy. Mrs. C., otherwise than as to headaches well, had a fall which injured her nose. The shock resulted in persistent headache without other cerebral trouble save complete loss of smell. Two years later she had, but only on going to sleep, a sense of horrible odors, which were fecal or animal, and most intense. This lasted several months, and then were added sounds of voices, which were at first vague, but at last accusative and soon were heard in the day. The case ended in melancholia with delirium of persecution, during which the trouble as to smell passed away.

CASE II.—A. C., a clever lad of exceptionably able and normal descent, became insane at eleven years of age, and was long maniacal and often homicidal. For some months before this outbreak—which was acute—he was troubled by seeing animals on his bed before he fell asleep. Opening his eyes routed them at once. The condition seemed to have none of the peculiarities of the night terrors of childhood.

[1] Baillarger: Annales Méd.-Psych., vols. 5 and 6.

I am tempted to add the brief notes of another lad's case, in which the same præ-somnic period was a time of singular disorder of mind. It is, of course, known to many of you that the bromides may in some persons (and notably in the young) occasion, like mercaptan, profound melancholy or maniacal tendencies which, in several instances in my experience, have been homicidal, or at least madly destructive.

The lad in question, an epileptic, was said to become homicidal from bromides. I was sceptical enough to test the matter. About the seventh day of using full doses of lithium bromide the trouble showed itself in the præ-somnic time, as visions of himself killing other children. It annoyed him greatly so that he strove to keep awake, but at last tired of the unnatural effort would fall asleep, with too brief an interval to allow of his being disturbed again. After several nights of like distress, the homicidal tendency broke out in abrupt and dangerous violence during the daytime.

There are epileptic cases in which inhibition of fits causes homicidal explosions which cease when the fit comes on, and do not recur for a time, whether bromides are continuously used or not. In the lad's case the fits did not lessen the tendencies to destroy or injure others. These lasted as long as the bromides were given. I think Echeverria mentions like cases.

There exists also, I may say, a group of cases (not in the books) in which the borderland of sleep is haunted by hallucinations for weeks or years without their ending of necessity in mental disease. But it is quite impossible for me here at this time to dwell on these interesting cases of which I have seen a few, and but a few. In no respect do they differ from the like cases reported by Baillarger in which insanity resulted, save in the fact that it did not. Voices were heard, or distressingly real visions seen during the præ-dormitium and at no other period. With time and due care these visions faded away. Two of the half-dozen I recall were hysterical women; two were men in busy affairs, and one, the worst, a woman of thirty-seven, was, I think, preserved from insanity by the loss of her ovaries.

The corresponding time, which comes after sleep and before full wakefulness, is also said by Baillarger to be troubled in some by hallucinations; but of this I have no experience, nor does he speak of it as common. Tuke also has reported cases.

There is yet another and stranger mental condition experienced, though rarely, in the præ-dormitium. This is a suddenly acquired and

sometimes persistent sensation of fear or terror without any sensory hallucination. For our emotional states we have usually a cause, or at least think we have, but what I now describe is an emotion without known parentage. Children may exhibit this continuous fear for some time after the scare of a dream, like the echo of an emotion the cause of which is over.

Sometimes in adults also this lasting sense of alarm is the product of a dream. The victim awakens and continues even for hours to feel the fear to which his dream gave rise. He is wide awake; lights the lamp; reads, or tries to; but is still fear-haunted, reason as he may. The patient who is liable to this fear may also be subject to attacks of pure fear without a dream cause, and arising in the time between waking and slumber. He has then no dream. Of a sudden, whilst half awake, the man is afraid. It is pure fear, such as the insane have at times. I append a case from my first paper. In it were both forms of the trouble here mentioned. The case is given, as stated by the sufferer, a scholarly, much overworked man, with no obvious habits, hereditations, or disorders to explain his condition. He says:

"About the year 1871 I was troubled with what I understand to be 'night-terrors,' but unlike any I have been able to hear of. Upon retiring I could generally tell whether or not I should have this trouble during the night. These premonitions were: A difficulty in breathing, not being able to draw a full breath, owing, as to me it seemed, to some obstruction in the lungs; intense nervousness; turning from side to side.

"I would fall asleep and have vivid dreams, and almost always upon the same subject, the purport of which was, that after long absence from home I returned and found that some one dear to me had become idiotic.

"The most painful attack of this kind occurred in 1872. That night I dreamed that after a long absence I returned, and, upon approaching the city, I saw upon a steamboat my aunt. She had become crazy in my absence and was under the charge of keepers. As I neared the boat to speak to her she leaped overboard and was drowned, and her body, with a fearful idiotic leer upon her face, floated past me so close that I could touch it. I awoke with a sudden start trembling from head to foot; and, although in a moment I realized that it was but a dream, yet the feeling of terror, instead of leaving me, rather increased. I was obliged to rise, light the gas, and leave the room and remain for several hours in an adjoining one. I then returned to bed and slept until morning; but the next evening, when it came time to retire, the recollections of the past night were so vivid, and the intensity of the mental suffering so clearly before my mind, that I could not force myself to retire. My reason told me that this was a foolish feeling and that I ought to

conquer it. But after a severe struggle reason gave place to this undefinable feeling of terror. That night, and for several nights afterward, although I was not addicted to drinking, I drank strong liquor until my senses were clouded, and this I did intentionally, otherwise I could not have retired.

"During the day-time, when thinking over this fearful attack, I concluded if it were given me the choice between passing one such night and being deaf, blind, or lame for life, I should choose the latter; nay, I felt that even death itself would be preferable to such another night. I have never since experienced such intense suffering, but have passed through it many times in lighter forms.

"This fall (1875) it took a different turn. Upon retiring I was unable to keep my eyes closed, because the moment I closed them a feeling akin to fright would cause me to open them.

"This was like, but incomparably less, than the dream-evolved terror. Nevertheless, it was bad enough. It did not come if sitting up I closed my eyes, but to lie down and close them was often enough; or, if the emotion did not then arise, it seemed to burst upon me just as I was conscious that sleep was near. These attacks were the worst. I was afraid—and of nothing. No reasoning helped me. As I am by nature, despite my professional life, hardy and courageous, I was rather ashamed of being fearful of nothing, knowing that in battle I had had no more fear than others, and none that disturbed me.

"After tossing endlessly for hours, I would at last sleep for an hour or more with the sense of sleeping illy. If I had a bad bout, I sometimes awakened with my mind not clear, and feeling as if I needed effort to steady it."

Despite these alarming symptoms, this gentleman got well after a summer in camp in Maine.

Others have described to me this state of fear in the pre-somnic condition. Says one:

"I have had, like others, nightmare, but this comes over me while I am quite conscious, and, of this I am sure, whilst yet capable of mental analysis, and just pleasantly drowsing. I simply and abruptly realize that I am afraid. I feel it coming. I am not paralyzed, as by nightmares. I can move. If I fully sit up, it is over; but, if I delay to do so, and it catches me, it stays on for a minute or two after I am completely awake and master of myself. I sometimes lie still with open eyes and seek to know why I fear, or reason it over, but nothing relieves me. The fear goes by degrees, but if at once I lie down again and close my eyes it comes back."

Another mental state, somewhat akin to this last (for fear and anxiety are near akin), also occupies the præ-dormitium. It is always an associate of bad sleep, or of insomnia, and consists in a series of

unreasonable fears and anxieties. I will let a sufferer tell his story as he wrote it for me:

He was fifty years old, of nervous, restless intelligence, anxious always, successful past the common, free from disease, endowed with a perfect stomach, and habitually insomnic. He called three hours a good sleep, and for years lived on this and an afternoon nap of an hour. Now and then, his restlessness got worse, and was the insomnia of over-vigilant and excited centres, which furnished a succession of anxieties, each in turn capable of inhibiting sleep. He says:

"This trouble haunts the time close to sleep. I lie down; am easy, and assured of sleep. Suddenly, I think, Is the gas turned off properly? I get up and look; return to bed; get up again, and so on. At last, I become anxious as to my son, aged six. Is he safe in bed? will he fall out? My wife goes to see, reassures me, and then I go myself, and go a dozen times. Next, it is the furnace, or the locks, or fear of fire, until, worn out, I am surprised by sleep. It seems as if this thing waits for me at the gates of sleep, and I can understand that just then one's fancies may run wild. But once awake, the thing goes on until I am ashamed of the demands made upon my wife, and, too, of my own folly. I know of others who have the same trouble, but never in the day season."

Sleep-numbness. Nocturnal Paresis or Paralysis.

As sleep-numbness, this disorder has become familiar since I described it in February, 1876, and later more fully in my book in 1882. Since then, Dr. Andrew H. Smith, in this country, and Dr. Saundby, in England, have written of it anew; the former as an undescribed neurosis, and the latter without full knowledge of what had already been written. Dr. Saundby thinks I appear to have recognized this condition, but is of opinion that the name I gave—nocturnal hemiplegia—in view of the very temporary loss of power, is not very appropriate. In fact, I described pronounced cases chiefly, and their duration had nothing to do with the nomenclature.

In general, functional day-numbness (as I and others have described it), whether neurotic, anæmic, gouty, diabetic, atonic, or asthenic, is apt enough to repeat itself at night in sleep. But there are people who never have day-numbness, and who are, nevertheless, liable to awaken with this interesting neurosis.

Definitions of it do not admit of sharp boundaries. It may be local, transient, a slight numb feeling, a faint tingling of the fingers, of a leg, or of one side; or else it may be intense, and present us with paresis

and real defects of the touch- and pain-sense. In another case it may show itself as an alarming monoplegia, or distinct hemiplegia, lasting a few moments, or growing worse during hours. Again, it may involve the whole body, but is then apt to be less severe than are the hemi- or mono-plegic forms. Yet, are they all akin. I have seen several of them in one person; at this time slight numbness, and at that, hemiplegia, alarmingly positive, with marked loss of power and with lessened sensibility. For Dr. Andrew A. Smith, waking-numbness is a paræsthesia at the exit from sleep, and "in this," he says, "there is nothing added, and nothing taken away." But these subjective states, which to-day are mere tingling or formication, may to-morrow deepen into the semblance of hemiplegia, with distinct loss of sensation. It is only a question of degree. The neurasthenic, the hysterical, the tobacco-poisoned, the gouty, the dyspeptic, are liable to awaken with numbness, tingling, dysæsthesia of a part, or of both hands, or of a side. It goes off in a few minutes. More rarely, there is distinct weakness of an arm or a side, with dysæsthesia, very rarely with complete loss of feeling. In every case where there is lessened or lost motility, the sense of touch is also interfered with. And so it is that we may have various degrees of disturbance from faint tingling to profound temporary dysæsthesia and defects of power from paresis, up to a brief simulation of paralysis. Were the worst of these simulations to last, they would be grave enough, and, in fact, it does sometimes happen that such states as I describe may even deepen in intensity after the patient is fully awake. In some patients they recur night after night, attack both hands or both sides on successive nights, or occur at intervals for years. I may add that I have sometimes seen this symptom in men apparently vigorous, and that it also occurs, now and then, in those who have multiple cerebral aneurisms or end-arteritis. I have seen it, too, over and over, in convalescent hemiplegics. They awaken with the palsy worse, a functional condition being for a time added to that which had an organic cause, or the sound side suffers, to their great alarm.

I give a physician's story of his attack:

"Excessive work, with double abuse of tobacco, had caused day-numbness, which troubled the ulnar territories most. One morning I awakened with transient numbness of the whole left hand with no true loss of touch. A week later, I had on awakening dysæsthesia, with pricking of the whole

right side, including face and tongue. I arose, found leg and arm weak, examined sensation in the finger-tips, and recognized the fact that with scissor-points applied to the finger-cushions, I could not be sure of them as two at one-third of an inch separation. I was about to send for a physician, when the sense of tingling becoming worse in the extremities, the dysæsthesia grew less, and in two hours I was as well as ever."

It is of interest that he soon after saw a case of waking-numbness in a tobacco-using patient, whom he confidently reassured, stating his own case. In fact, however, the patient owed his numbness to unrecognized diabetes, and it ended in gangrene.

The history of numbness in all its grades points to a central origin, but that it may arise otherwise is also clear. In some cases of neuritis I have seen night-numbness as a first, a transient, and a repeated symptom preceding the pain by several days.

Some years ago, whilst writing my book on *Nerve Lesions*, I froze my right ulnar nerve with alcohol at 0° F. For ten days afterward I had more or less discomfort, and even acute pain, but especially, several times, a positive numbness of the ulnar territory with which I awakened. It passed off with friction in an hour, but was much worse than the occasional day-numbness, which my experiment created. Yet, for a time, it was purely a waking symptom and faded swiftly. The more profound examples of hemi-anæsthesia with paresis as post-somnic states are, I think, most apt to involve the right hemisphere. I recall no instance of aphasic accompaniments in the rarer left brain disturbances. In their varieties these symptoms probably represent functional irritations or inhibitions of quite various parts of the brain; unlike their hysteric related states, they are felt in the face as well as below it, and are clinically of kin to the functional anæsthesias and pareses of certain migraines.

In some cases, notably in hysteria, waking-numbness is associated with pain in the parts affected, or there are also paræsthetic expressions, as sense of constriction, elongation, etc. At times, sensation resembles the furious formication of aconite-poisoning.

In Dr. Ormerod's paper[1] on numbness (which is the best of the English essays) he speaks of the pain suffered by some of his cases, and as to this he is quite correct. In fact, there are those who awake with pain in arm or leg, or both, so intense as to make the accompa-

[1] St. Bartholomew's Hospital Reports, 1883.

nying numbness seem of little moment. Pain as an accompaniment is also mentioned by Sinkler, but not as being severe; whereas, it is in some instances of waking-numbness very great. I add two or three cases. The first, again that of a physician, I leave as he wrote it. He says:

"The preparative causes of my present state were excess in tobacco, and a practice which left me no rest. Then came a domestic calamity, and I broke down with distress in the occiput, and an amount of suddenly developed physical feebleness which annoyed me. I could not walk up three flights of stairs without resting. My heart became rapid—90 to 100; my temperature 97.4°, and at night 98°. I was struck, too, with the weakness of my bladder, the urine merely falling as it emerged. All this was in December. In February I began to have night-numbness, and would wake in the night with all four limbs numb. The worst attacks were those at waking in the morning late. Early in the night the symptoms were less notable, or else affected one limb only, or one side. At times it was mere tingling; at others, positive lessening of sensibility to touch and pain. When this was the case, the limbs were for a time paretic; on one occasion so much so that I fell on getting out of bed. The trouble has not lasted usually more than thirty minutes, but went with great tingling, as of a limb asleep, as is said. After March, I began at times to have numbness in daytime, but rarely so intense as to disturb feeling. The tingling occasionally affected my whole surface, and was apt to begin around the mouth."

In this case the symptoms were neurasthenic, and absolutely no other cause could be assigned for it. Recovery was complete. The next cases are from Dr. Féré,[1] and are certainly hysterical and of great interest.

"*Nocturnal Paralysis.*—Mrs. V. came to consult me, for the first time, at the Saltpêtrière on the 12th of January, 1885. She was accompanied by her mother, who was over sixty years of age, but still very agile, and looked much younger than she really was. The mother had a painful ovarian spot with slight anæsthesia on the left side. Although the menses had ceased nine years previously, she had been subject to migraine, with attacks of melancholia, and occasionally convulsive fits. The father, who had been a drunkard and profligate, had quitted the house twenty years before, and no one knew what had become of him. A brother of the father had died in prison while undergoing confinement for swindling. Mrs. V. had two sisters born after her. The one had died of convulsions connected with teething at the age of eighteen months; the other died of convulsions when only six months old.

[1] Brain, October, 1889.

"Mrs. V. had been a precocious child both physically and mentally. She had walked and spoken at a very early age, and had learned very rapidly at school. She has never had convulsions nor tic, but from the age of six has suffered from frequent migraine, followed by vomiting, and during her whole life her sleep has been troubled by nocturnal terrors and nightmare. Menses began when she was twelve. At the age of seventeen she had attacks of chorea in consequence of worry. This lasted three months, and chiefly affected the left side. At nineteen she was married, and had her first child, a boy, when twenty-three. This child died of convulsions on the eighth day. In the following year she was delivered of a child stillborn. During her pregnancy she had anorexia and vomiting, which ceased spontaneously in the fourth month.

"Since her chorea Mrs. V. has always enjoyed good health, and has had no distinctive nervous outbreaks until about three years ago. At the time her husband died she suffered severe pecuniary loss. This induced insomnia, followed by loss of appetite and emaciation. By hard work her health became pretty good. About two months ago she had a very abundant metrorrhage. A few days after this mishap she began to feel constrictive pains in the head, extending over the whole of the cranium, but predominating in the postero-inferior region, which appeared, moreover, to be the seat of a constant pressure. From time to time she heard a cracking noise in the back part of the neck, which resounded in the occipital region of the skull. At nightfall she was seized by painful fancies, of ruin for her mother and her daughter, of illness for all her friends; at the same time she was a prey to unusual pusillanimity and indecision. Her sleep was disturbed by dreadful nightmares. She was widely awake toward six in the morning, but was incapable of making any movement. She suffered from distention of the bladder, but could not even think of getting up. Her limbs seemed numb to her, and as if wrapped in cotton. She appeared to know the position of her extremities only, and it seemed to her as if the greater part of each limb was wanting, and her hands and feet had been brought up close to her body. The sensation is analogous to that experienced by amputated persons, who say they feel only the extremity of the absent limb. She could make no movement whatever. When daylight was admitted a sensation of numbness and pricking gradually appeared in the extremities of the fingers and toes. These sensations, occasionally very painful, preceded the return of ability to move. About eight o'clock the patient could get out of bed, maintain herself in a standing position, and make movements of the arms. The complicated movements of the fingers, however, remained almost impossible. She was thus incapable of fastening her clothes, or of taking up a pin. When she had moved her arms and had been rubbed a little, her fingers became more supple. From the time she awoke until the nearly complete restoration of movement took in general about three hours. One day, when left in a dark room until about ten o'clock, she was found in the same helpless condition. Movements of the head and neck and of articulation were not affected. On examination

no modification of the external aspect of the limbs could be determined. She suffered pain in the region of the left ovary, and had slight anæsthesia on the same side. The contraction of the field of vision was tolerably extensive, and the patient was insensible to violet rays in the left eye. The iris of the left eye is of a deeper brown, and the pupil not so large as on the other side. Under the influence of bitters, iron, bromide of potassium, and hydrotherapy combined with static electricity, all these symptoms disappeared in the space of three weeks, with the exception of pain over the ovary and the hemianæsthesia.

"Case of Mrs. P. She complained of sudden shocks in the head which awoke her abruptly. These occurred by night four or five times. Finally came other troubles which raised her inquietude to a climax. When her sleep became broken, toward four or five o'clock in the morning, she found that she could not move any of her limbs. This general helplessness did not last very long, for after a few minutes of effort she recovered power of movement in her right hand and foot, but for the limbs on the left side prolonged friction was necessary. This paresis was accompanied chiefly on the left side by a sensation of painful numbness and pricking. The hand especially was quite cold, and the fingers appeared to diminish in volume, the rings hanging quite loosely.

"This helplessness, which at first persisted only a few minutes, was in a month's time prolonged for an hour or more. The patient could not raise herself out of bed until some one had opened the windows widely and rubbed her energetically. Even then she would remain for many hours incapable of making any delicate movements or even of simply fastening her dress. When the paralysis was at its maximum the patient declared that she was no longer conscious of the existence of her own body, that she was, to use her own words, a *pur esprit*.

"Under the influence of cold douches repeated twice a day at regular hours and a tonic treatment of iron, nux vomica, and arsenic, with bromide of potassium given every evening in moderate doses—one to three grammes—her condition rapidly improved. The paralytic numbness on waking diminished at once in duration and then in intensity. At the end of fifteen days it had almost completely vanished. The sensations of shocks in the head, which had caused the patient to awake, disappeared in turn. The anorexia and pains resisted longer; at the end of six weeks the pain in the tendo-Achillis still persisted, but in time it too disappeared."

It is very clear that a vast range of disease or disorder seems capable of causing night-palsy in its variety of degree. As concerns the cause, there is difference of opinion, from those who look upon it as due to rare somnic phases of the vessels of the brain to those who see in it, like Féré,[1] a deficiency of physiological excitation—"paralysis

[1] Brain, October, 1889.

from irritation." Reflex it surely is at times, and local or peripheral rarely.

As to treatment, it is folly to discuss it. It means anything from the treatment of neurosis to that of gout, renal insufficiency, diabetes, and what not.

SLEEP-PTOSIS.

Another form of annoyance to which I have time to give but a passing notice is the ptosis of sleep. Of this I nowhere find mention. The patient wakens with palsied lids; lifted, they fall again. In some instances it lasts a few minutes or longer, or else before the disorder repairs itself sleep returns, and at morning the patient has it anew, or awakens without it.

I append two cases which came under the care of Dr. de Schweinitz:

One was that of a woman, aged sixty-five years, as I understand in good health and not hysterical. There was some irritation of the lids and slight general conjunctivitis, also there was a slowly ripening double cataract. As the vision was still fairly good she read much, and if she used her eyes freely in the evening was sure to experience the following trouble: waking in the night, she found that her eyes could not be opened by the will. After they were forcibly opened they remained controllable by volition until after sleeping again, when the same phenomena recurred.

The notes are not complete, and although she describes the ptosis as spastic, it seems to me to have been only a night-palsy, of temporary duration. A somewhat similar case was observed at my clinic a few weeks ago, and I have seen a number of them.

J. C., aged twenty-nine years, a widow, was in absolute health after a childless marriage of four years. Soon after her husband's death she began to suffer with sexual dreams, and later, with these and hemi-anæsthesia of decisive type without loss of power. A year from the beginning of her widowhood she awakened thrice in one night with paralysis of the lids. There were no eye troubles, but the lids as she awakened could not be lifted at will. When raised by a finger they fell flaccid, and only by degrees, in an hour, recovered tone. This trouble, unknown to the day, continued for many months, and, indeed, disappeared only when this and all the other symptoms were promptly dispersed by a second marriage.

In another case the ptosis was like the emotional spastic ptosis of hysteria we see in waking hours, and well known to neurologists:

A woman, aged sixty-seven years, in good general health, but full of notions; for fourteen years has been the subject of stubborn retinal asthe-

nopia; the eye-grounds are, however, healthy; there is a considerable degree of hypermetropia and astigmatism, together with insufficiency of the internal straight muscles. Each night, for a long time, usually between twelve and two, she has been awakened over and over by a feeling that her eyes have closed spasmodically. She arises, forces them open with difficulty, and bathes them, "because the lids feel as if they were sticky and would glue fast." The phenomena repeat themselves, but never by day.

This case proved, I believe, very obstinate, and is only notable because it is of nocturnal origin alone.

Sleep-pain.

I add a few words as to what is, for rarity, a medical curiosity. I find on my note-book some half-dozen cases of pain in the legs, never known to the day, and needing sleep as a conditioning factor. Whenever I have spoken to a patient of this obscure disorder he is apt, if intelligent, to say: "No, not pain; it is distress." It occurs in middle life, or later, and, without previous disease, comes on slowly. In one case it followed a typhoid fever. The cases are alike. A man is well in the day; walks, works, does as do others. Some time after he falls asleep he is awakened by aching in the legs, from the sole half-way to the knees. There is but one remedy—motion. He rises, walks for ten minutes, is eased, goes to sleep, and in an hour or two awakens to feel the distress and repeat the relieving exercise. There is no unusual heat or cold; nor any abnormal appearance. The matter is unpleasantly simple, and there is no clue to a cause.

The case I now quote from my note-book I saw two years ago. It is typical of a rare condition:

J. C., aged fifty-eight years, farmer, Kentucky; married; two healthy children; himself of sound breed. Has worked hard, and has never known a serious illness. No malaria. In October, 1886, consulted me; I found him a man weighing 180 pounds; height six feet, vigorous, with soft arteries; heart beating seventy-six, and perfect. He has no piles, hernia, or varicose veins. Eats well; is in all ways moderate and regular; does not smoke, and does not chew tobacco. All the reflexes are normal; the heart and arterial tension healthy. No organ is diseased; can work all day; no tenderness in nerve tracts.

About four years ago be began to wake now and then with distress in the legs. During the next year this got worse, and now is a source of extreme suffering and of disablity, because of the loss of sleep it causes. Within an hour after bedtime he awakens in what he calls torment. From the knee to

toes his legs ache, without throb or sharp pain. He rises, walks until weary, goes to bed, sleeps, and wakes to the same pain or distress. Again walks, and so on, until day brings relief.

At the infirmary we learned that in the attacks the legs remain normal as to temperature, reflexes, and electrical conditions, as well as to sensation. No remedies were of the least use, except morphine, and I advised its steady use in despair as to other means.

Sensory Shocks.

Another phenomenon of sleep, or its borders, and also in the sensory sphere, I first described as sensory shocks or discharges.[1] This is a more rare, but also a more interesting disorder than numbness. Except my own paper and a small book by a homœopathic practitioner,[2] I know of no literature on the subject, beyond a few lines of remark on my paper in an essay of Hughlings Jackson's. Nevertheless, the subject is still of interest.

All the disturbing phenomena of sleep at some time represent themselves, more or less well, in our waking hours. But sensory shocks are, of all disorders of sleep, the most rare in daytime.

In the præ-somnic state of fading sensation, but never on waking from sleep, the patient has in his head a sudden and violent sensation, and the forms it assumes may be classified thus:

I. *In the sphere of general sensation.*—He feels as if struck, or as if he had a shock like that which a sudden arrest of motion causes; or, it is a feeling of rending, or a bolt driven through the head. These are the most common.

II. *Auditory.*—A loud noise, like that of a pistol-shot, or of the crash of broken glass, or as of a bell, or a wire sharply twanged.

III. *Visual.*—A flash of light.

IV. *Olfactory.*—Sudden sense of an odor.

V. I doubtfully add what I may call *emotional discharges.* These are always mere abrupt sense of fear, sometimes *preceding* the sensory shock, and sometimes *following.*

What happens is usually this: A man going to sleep, but still quite conscious, and able to observe, feels suddenly a shock in the head. It

[1] Virginia Med. Journal, op. cit. Lectures on Nervous Diseases, op. cit.

[2] In Dr. W. S. Searle's book he described sleep-shocks as a neurosis hitherto unmentioned in medicine, and was evidently unaware that I had already and fully delineated its peculiarities.

seems mechanical, as of a blow, or noise, or else of both; and also there may be added a flash of light, vivid or like the soft summer lightning along an evening horizon. The intensity of these phenomena may be appalling, and even those who are used to them greatly dread their return. The first experience is sometimes most alarming.

In many cases there is an aura. A physician, who came to me on account of these attacks, first called my attention to this. While waiting for sleep he became aware of an indescribable something, which rose from the feet and hands, and taking eight or ten seconds to reach the head, there ended in a sound like the crash of glass houses breaking in a hail-storm, with a vivid flash of yellow light, leaving him for a moment dazed, but able at once to rise, or to think. Such is the usual account given of this aura. It never varies, save that it may rise only from the belly.

All sufferers, or nearly all, who have aura, say they can stop the attack by turning over, or sitting up, or even by opening the eyes. A friend, who had this trouble owing to tobacco, says: "The shocks were of two kinds; one as if I received in the brain a thump, and one as if a pistol-shot occurred *in* the head. I had for a long while no idea that the pleasant mistress, Nicotia, was disturbing my internal economy. At last, before learning this, I got used to these abnormal things, and would lie still and feel what you call the aura. By the way, it is not at all like an air, but like the surging upward of something more positive. When it gets to the neck I am gone; the explosion occurs. Below that, I can avert the wretched thing either by rising or rolling over, for it never comes except I be on my right side. No mental effort suffices to check it."

I have notes of two males who suffer only if on the right side, but I have not always made inquiries as to this point.

The aura is said at times to be like a tingling, or else is described as an upward surge of indescribable nature, and at times only rises from the epigastrium. Often there is none. Once felt in any case, it usually continues in some form to precede all future attacks.

There is another form of warning, which patients only succeed in describing as a state of brain which foretells the shock. I have heard this called a humming, or buzzing, within the head.

Hysterical women are often quite unable to stop the shocks, or the aura is too swift to be a timely warning. It is rare to find any grave

result. A few people become vertiginous, but not severely. At first, alarm causes emotional scare and a quickened heart, and in some few, even when repetition has lessened the terror of the shocks, they cause a more prolonged palpitation of the heart. In a few minutes, when again sleep is near, comes another shock, or there are a number of slight attacks, as of a bell, or a guitar string twanged, and by degrees fading away; visual discharges less commonly recur in this manner.

I have over and over met with cases of sensory shock in the day-time, but only in the hysterical. In some few people they occur during sleep, and awaken the patient, but their habitual place is in the præ-dormitium.

All of these curious outbreaks represent, as a rule, the "coarse stuff" of sensory product, or something near it. There is sense of shock alone, or this in succession with noise or light, or both. The noise is rarely comparable as to note of music, or as to a bell, or a string vibrating. And so also of the optic explosion. There is light, violet or pale yellow. No visions appear; no voice calls. It is "primary sensory *stuff*," and no more. In the one case connected with smell, the patient had an epigastric aura and smelt bananas. She had anosmia.

These singular symptoms are found in some neurasthenics, in hysteria, and most often in man as a result of over-use of tobacco-smoking. I first knew of it in my own case, during an attack of neurasthenia, many years ago, and soon learned that it was immediately due to my cigar. I can assure you that one's first acquaintance with it is most terrifying. Bromides and strychnine control it, as I have elsewhere stated.

I pointed out years ago the interesting resemblance of these innocent attacks to epilepsy, and Hughlings Jackson has also noted the fact. Bennett,[1] in a paper on the "Sensory Auras of Epilepsy," describes cases in which the signal-aura consisted simply of crude sensation, such as tingle or pain, which he calls sensory epilepsy. These are sensory cortex centre explosions, with consciousness and without spasm.

Next to touch-aura in epilepsy, comes most often optic-aura—sense of light, or definite color with form. In the præ-somnic explosions the visual phenomena are still more crude than in these. But in epilepsy,

[1] Lancet, April, 1889.

auditory auras are rare, and gustatory and olfactory auras still more uncommon. Certainly, many epileptics who have auras at times experience these alone. The patient feels as if struck a sharp blow, or perceives a noise in the head, or sees red fire, or a flash of light, and has no fit. Or else he has a subjective taste, or, like a physician I know, smells human ordure for half an hour, and may fail to have the usual sequent spasms. These are very like such præ-somnic shocks as give us in their completest form a sense of tingling, etc., which, rising, ends in a more abrupt sensory discharge, as of a sensation of shock, light, or sound, or these variously combined, as does also occur in epilepsy sometimes, as I have seen. Again, too, in the subjects of sleep-shocks are found those who have in the præ-somnic state conditions of terror or daze, which are brief, and represent explosions in the intellectual or emotional sphere, like the dazed or dreaming states of some epileptics. And, curiously enough, this, too, may in our illustrative shocks be preceded by a sensory aura of one or two senses.

In epilepsy with auras, we may have, first, an aura, *i. e.*, a sensory discharge, usually simple tingling, and then a sense of flash or sound, or of light and sound together; the centres being, one may notice, in juxtaposition.[1]

In præ-somnic discharges the discharge is simple, or preceded by tingling—true aura—vague epigastric sensations. The analogy to epilepsy is closer when, as often occurs, an uncontrollable jerk or flap of the whole body ends the attack. With motor centres pathologically over-excited, or capable of being morbidly over-charged, widespread epileptic motor phenomena may follow the sensory discharges, and in early cases of sensory explosions, hysterical convulsions of mild type may result, and thus bring us still more near to the sequential chain of epileptic conditions. On the other hand, epilepsy is not a disorder which haunts the præ-somnic time. However close the apparent analogy, there must be a wide difference between it and these sensory explosions.

Lastly, there is a form, which I have seen but twice, in which we have an aura—a flash-shock or sound—and a sense of pain darting down the cervical spine, and then along both arms to the finger-ends.

[1] Angular Gyrus and Superior Temporo-sphenoidal Convolution.

SLEEP-JERKS. CHOREA.

In the motor sphere are certain disorders which trouble the sleep, or præ-somnic state in hysteria or neurasthenia, and which are only distinguished from phenomena found in health by their excesses. We all have had the common experience of a sudden jerk of the body as we were falling asleep. This, in certain cases, is exaggerated as to degree and number, and is also found in the sleep. I have seen many cases in which, scores of times in each night, the sleeper was awakened by a violent motor discharge of every muscle at once. In others the jerks are of arms or legs only. Probably, what are known as "foot fidgets," which oblige the sufferer to move in order to get rid of an ever-recurring sense of unease, are, too, of like parentage, and are to be seen in all degrees of intensity. I was lately consulted by a Western banker, æt. forty-six, to appearance well. No organ was diseased. He had passed successfully through a time of great financial trouble, and in its midst his wife fell ill. After she recovered, he began to be a poor sleeper, and exhibited in turn a variety of sleep troubles. He had a mild form of shock—*i. e.*, light and noise. Disuse of tobacco aided these and improved his sleep, but, somewhat later, he began to have jerking in sleep. An arm, or the leg, or the body was violently moved, so as to wake him up, and this, he declared, took place countless times in the night. Still later, he lay awake with a horrible unease in the legs. He moved about and got ease. If he lay still, he had to move again.

These motor discharges at times assume, through their frequency and severity, such importance as to affect health by the destruction of sleep which they occasion. An instance is given in my book of a woman weighing two hundred pounds, who spent her nights in a series of motor explosions so vigorous as at times to break the bed-slats. She has told me that she believes herself to have had as many as a hundred in a night; the whole body moving violently in sleep with a jerk like the leaps of a dying fish.

A different form of unease is seen in children, who nevertheless seem to be well. Their sleep remains unbroken, but they roll over, twist, turn, wriggle, and continue to do so for hours. Possibly, they are dreaming, but of this there is often no evidence, and they are not affected in health by this extraordinary restlessness, which may remain

as the habit of a year or more. I have over and over watched these little ones in a sleep which permitted them to roll over and bend the body and move the limbs, until it seemed scarcely possible that they could remain through it all in a state of slumber. A little pause might follow and then another period of nearly constant movement.

In adults this extreme of restlessness is very uncommon, and means more than in childhood. Of the hysterical sleepers much might be said, but in them this form of activity during sleep is seen at times.

In the singular ataxia of hysteria, which I described a few years ago, the early stages of the disorder are apt to exhibit on waking an ataxic condition, which becomes increasingly worse, and at last continuous through the waking hours. More commonly the ataxia comes on by degrees, and only in the day.

Lastly, in relation to the motor sphere are the rare examples of chorea seen only for a time on waking from sleep, of which elsewhere I find no mention. As regards this, I may remark that some early ataxics, and some neurasthenics, are apt to be unsure of their movements for a little while after waking.

Chorea in the Post-dormitium.

A. B., aged fourteen years, menstruating regularly; somewhat anæmic, but in other respects healthy.

Last spring, in the month of March, she was attacked by a singular form of chorea. She had this trouble at no time except in sleep and on waking from sleep; on either occasion it was in attacks which did not endure very long. Her mother, who frequently watched her in the night, said that three or four times in each night she became restless, kicked off the covers, and began to move her hands, slowly flexing or extending them until at last the arms also moving, a general choreal movement ensued, which, at the same time, affected the legs, arms, and body, but never the face. The attacks affected her almost always when she awoke from sleep, during the time she was suffering from this disorder. In the warm summer weather it disappeared. It has returned again recently. She has been under my care for some time, so that I have had an opportunity of seeing that she is a person in perfect health with no organic disease about her on which I can lay a finger. The attacks in the night are very rare, but she scarcely passes a morning without waking in this choreal condition. The spasm lasts from a half to three-quarters of an hour, and by degrees fades away. She apparently has no control over her movements, and in this respect they differ from ordinary choreas, except of the worst kind. This special manifestation of chorea must be extremely rare. In an experience of many years (in which I have

seen a multitude of cases) I recall but three or four of this character. It is mostly confined to sleep, or to the awaking state, not apparently existing during the day. I have no hesitation, however, in classing it as chorea, because it readily yields to the treatment which is given to choreal cases, and because, in one instance, it occurred in a child who had had two previous attacks of chorea.

This instance is, perhaps, worth relating:

C. J., a clever little boy of about twelve years—not very strong, nor very active-minded; never rheumatic, heart normal; not very fond of out-door sports, and somewhat anæmic—was attacked one spring with chorea, from which after two months he recovered. The following spring he was attacked again; this attack lasted seven weeks, after which he again got well. The next spring it was replaced by the peculiar form of post-somnic chorea of which I have spoken. He had absolutely no chorea during the day. He usually woke up about half-past seven in the morning with choreoid movements of the hands; both sides were alike affected. They were not in character like those of the girl mentioned above, and were more distinctly under control. For half an hour, however, he could not pick up anything without dropping it. What struck me with him, also, was that the face was not concerned in any way; nor did it affect either leg. Sensation did not appear to be affected, and he was relieved by the ordinary arsenical treatment and cold douches.

Tonic Spasm

is another rare trouble born of slumber, and lasting after it. I quote the only cases my note-books afford:

Mr. J. C., aged forty-five years, merchant, had syphilis and distinct secondaries at the age of twenty-three years. Was well at the time of the malady about to be mentioned—that is, he had no perceptible organic trouble.

About four years ago he began in the early mornings to wake up with rigidity of the legs. This was so extreme that it was impossible for him to bend the ankles or knees, or to elevate the knees at all. If they were lifted by another with difficulty, they fell slowly in extension. It was truly, therefore, a distinct tonic spasm. If at that time he had excess or defects of knee-jerk I cannot learn.

This state continued to show itself for over two years, occurring at intervals; sometimes taking place every morning for a week, then lapsing for a month, but never existing at other times than when he came out of sleep. If he woke up in the night, he occasionally had this same condition; but this was far more rare. It was commonly a morning affliction, and lasted for a few minutes, or at most an hour.

After a certain length of time the symptoms disappeared, but, owing to a bout of drinking, they renewed themselves. Again he got well, and a period of two years elapsed without further trouble. He had begun to have vertigo,

followed by difficulty in controlling his water, and this was followed by incoordination, and the entire range of ataxic troubles to which he is now a victim.

I quote another case as a still more remarkable example of rigidity developed in sleep and continuing for a time after waking:

The patient was a man in good circumstances, aged forty-five. He was in the habit occasionally of awakening in the middle of the night with rigidity of the legs. The limbs were violently extended, the feet being so completely flexed as to be straight with the line of the legs. It was almost impossible to lift the legs without lifting the whole trunk, so tightly were the muscles contracted, and so rigid was the whole mass, including the intra-pelvic group. This did not seem to be due to anything in the way of specific disease, nor to bad habits. The man had no disease to which I could relate it, except that he had been for many years a dweller in the lower part of the city, and had had attacks of ague year after year, and one very severe attack of remittent fever. Beyond this there was nothing, and these symptoms had long since disappeared; neither spleen nor liver was enlarged; nor, at the time of the rigidity, was he suffering from any malarial difficulties. The kidneys, heart, and lungs were alike sound, and to this day I remain puzzled as to the causation of this very peculiar malady. I saw him several times, because he used frequently to ring me up in the night in order that I might witness this affection, which was painful from the intensity of the contraction of the muscles. I have heard him scream from what he described as "positive agony." Indeed, nothing relieved it except full hypodermics of morphine, under which, slowly, within a couple of hours, the muscles would relax, but always, after an attack would remain extremely sore for days together. He finally ceased to suffer.

I have seen a disorder of the same kind, or something similar, in hysterical women, but even among them it is very rare, and it is not necessary for me to go into details. The state is merely an hysterical curiosity. I mention it to complete the list of peculiar cases which I select from the odds and ends of my note-books.

Respiratory Failure in Sleep.

I conclude this study of "The Disorders of Sleep" by calling attention to one of very great interest. I believe that it has been described by the late Professor Samuel Jackson, but I have been unable to find his paper, which is not in the catalogue of the army medical library. I recall, however, hearing him speak of cases in his lectures.

Where, for some reason, the respiratory centres are diseased or disordered, a man may possess enough ganglionic energy to carry on

breathing well, while the will can supplement the automatic activity of the lower centres. But in sleep, these being not quite competent, and volition off guard, there ensues a gradual failure of respiration, and the man awakens with a sense of impending suffocation. This is not to be confounded with the hysterical sleep symptom of sense of suffocation, which is probably closer to the phenomenon of nightmares, and is followed by, or associated with, fear, and is soon lost on awakening.

In the cases I refer to, the symptom is sometimes a signal of dangerous meaning. I have met with it in extreme neurasthenia, but in worse forms in locomotor ataxia in its paralytic stage. I have never seen it in labio-glossal lingual paralysis, where it would seem likely to occur. In ataxia it may be due to sudden incompetence of laryngeal muscles, which are liable, late in ataxia, to become paralyzed. Usually, however, it seems to be a failure of the chest and diaphragmatic movements.

Mr. C., aged fifty-six years, had posterior sclerosis, but gave no evidence in the day of respiratory incompetence, although he was distinctly far in the paralytic state. When in deep sleep, he began to breathe less and less deeply, and at last, for a few seconds, not to breathe at all. At this moment he moved, twitched, and at last awakened with evidences of commencing apnœa in the color of the lips, tongue, and nails.

When awake a few voluntary efforts to respire relieved him. These attacks became at last so frequent and perilous, that a nurse sat by his bed and awakened him as soon as he began to breathe less and less deeply.

As time went on, the trouble increased, and whenever he fell asleep, respiration ceased abruptly. He was finally worn out with loss of sleep, and died suddenly in one of these onsets of respiratory failure. No post-mortem could be obtained.

I have seen two other cases, but none so remarkable as that I have briefly related. But on the morning after I wrote these lines, I saw a case in an ataxic not yet in the paralytic stage. Just at the moment of falling asleep, he feels a sense of suffocation, fails to respire, and in great alarm sits up. These attacks probably differ somewhat from those of sleep.

The type differs from that of the ordinary Cheyne-Stokes respiration, being merely, and only in sleep, a gradual failure to inhale—a less and less deep inspiration; but no sequence of rapid breaths ending in dyspnœa.

DISORDERS OF SLEEP: INSOMNIA.

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In studying the disorders of sleep I have confined myself to insomnia, partly because of the inherent difficulties and extent of the subject; partly as my material is so crude and undigested as regards the more abstruse phenomena, that I have left them to the wider experience of our referee, who, as Schiller said of Goethe, need only shake the limbs to have the mature fruit of a ripe knowledge fall in abundance. I find that nearly every physiologist has attempted some theory of sleep, without adding materially to our knowledge why or how we sleep, although the result has been to indicate many of the phenomena which occur while we sleep. Duval calls the suspension of cerebral activity in sleep a function of the brain—the passive condition following the active as regularly as an interval of rest succeeds the heart-beats. Lasègue speaks of the appetite for sleep, which others liken to hunger and thirst. We certainly explain nothing if we say that drowsiness arises from exhaustion of the activity of the brain-cells, or, with Landois, that sleep is due to the using up of the potential energy, especially in the central nervous system, making restoration of energy necessary. Nor do we go much farther in speculating on the existence of a sleep-centre in the brain. The chemical theory of Obersteiner and Preyer, that the acid waste-products produced by the activity of the brain finally reach a degree sufficient to arrest it and thereby cause sleep until their elimination, is only less satisfactory than Pflüger's view, based upon experiments by Pettenkofer and Voit, and the oxygen hypothesis. The lessened amount of blood in the brain during sleep, as shown by the plethysmograph and otherwise, like the slowed pulse and respiration, cannot be held to be an antecedent cause of sleep, and the increased alkalinity of the blood

from greater proportionate elimination of carbonic acid naturally explains nothing in that direction.

What happens in the brain when we sleep is described from a psychological point of view by Mercier,[1] following Hughlings Jackson and Herbert Spencer, as follows: The molecules that were shifted into new positions are still oscillating from the shock. Those that were only slightly disturbed are returning to their original positions; those that were profoundly moved are settling in their new places, presenting more stable attitudes to the action of surrounding forces. Regions that perhaps after long repose were disturbed in the day still continue to emit a mild reverberation of their discharge, and the passage of currents at random through the highest regions under the sole influence of irregularities of pressure, and without guidance from external impressions, has its mental counterpart in the phenomena of dreams.

On the pathology or psychology of wakefulness, I have nothing with which I feel justified in taking the time of the Association. Indeed, the subject in general is difficult of concise treatment or scientific arrangement, and analysis of cases proves unsatisfactory. An examination of my records indicates the following causes or conditions of insomnia, each one of which might be easily expanded into an essay by itself:

1. The perverse habit of sleeplessness, a result of years, perhaps generations, of misuse of body and brain, I hardly need mention; and scarcely more the habits formed to induce sleep, as regards light and darkness, noise or stillness, idiosyncrasies of position, temperature, etc. These people are always amazed to see how much can be done to form the habit of sleep; what fair sleepers they may finally become by the proper kind of training.

2. Insomnia from external causes, through the various senses, excluding habit, naturally includes the obvious natural sensations of heat, cold, pain, hunger, light, noise, etc.

3. Excessive intellectual or emotional activity, including strain, excitement—pleasurable or distressing—grief, fear, worry, anxiety, etc., if sufficiently intense or prolonged. Naturally, as elsewhere, excess is only a relative term in different individuals, or in the same individual under different conditions. With the forced overaction of

[1] The Nervous System and the Brain, p. 374.

the brain the nervous discharge, as time goes on, requires greater stimulation until such a degree of exhaustion and instability is produced that any natural function, even rest or sleep, for a time becomes fragmentary, insufficient, or almost impossible.

4. Of the reflex causes of insomnia, indigestion, gastric or intestinal, is by far the most common, and it may fail to be indicated in any of the usual ways. As in persistent headache, a cause may be discovered only after repeated experiment in withdrawing one article of food after another, until the offending one is found. Genito-urinary and pelvic disorders come next, with unlimited possibilities thereafter.

5. The traumatic antecedents of insomnia are purely physical, from a sudden fall or blow, for instance; psychical, as in violent mental shock; or both, such as occurs in railroad accidents, etc. In these cases there is evidently a wide difference in the degrees of injury to the brain. The impression may be transient and resulting in restoration to healthy function. It may consist, in almost any degree, slight or great, of disarranging, decomposing, decompounding of cerebral molecules and cells, possibly of association-tracts, sometimes resulting, as Westphal held, in minute patches of sclerosis.

6. The auto-toxic sources of insomnia in acute diseases need only bare mention; in chronic disease, gout, lithæmia, rheumatism, tuberculosis, syphilis, malaria, leukæmia, and chronic nephritis, there is a similar cause independent of any cerebral exhaustion or impaired nutrition which may be produced by them. Probably much at least of the sleeplessness from habitual constipation belongs in this category. I have often found persistent wakefulness an early symptom of some general disease, especially acute rheumatism and arterio-sclerosis, many months before an absolute diagnosis became possible.

The habitual excessive use of tea, coffee, tobacco, alcohol, morphine, chloral-hydrate, bromides, cocaine, or other drugs, is a fruitful toxic source of insomnia. Chronic poisoning from arsenic and from lead, perhaps often by leading to vaso-motor disorders, degenerative disease, and arterio-sclerosis, without the usual symptoms, must not be overlooked.

7. Exhaustion from wasting diseases and enfeebling conditions, pulmonary consumption, anæmia, starving, profuse and repeated hemorrhages, sexual excesses, impaired nutrition of the brain from either deteriorated quality or diminished quantity of its blood-supply.

8. Of vascular origin, from hepatic disease, producing venous stasis, from cardiac and renal diseases with increased vascular tension, from asthma and hypertrophied or dilated heart, producing cerebral hyperæmia, anæmia or venous engorgement, from arterio-sclerosis, especially that of old age. Exaggerated or insufficient blood-supply to the brain, venous stasis, increased vascular tension, may also arise from syphilitic disease of the bloodvessels, or from disorders of digestion or of the portal system, and be as fertile a source of insomnia as the disturbances in the circulation in women at the menopause and other critical periods in life, including puberty, pregnancy, childbirth, and lactation. Here also there may be disordered innervation, as well as disturbed vascular tension and, perhaps, nutrition, such as we often see in less degree during menstruation.

9. *Vaso-motor.*—In that marvel of scientific research and profound medical knowledge, Meynert's *Erkrankungen des Vorderhirns*, the author repeats a statement previously made by him, that the nutrition and the *Erregbarkeitsverhältnisse* of the brain depend upon its relative weight, as compared with the weight of the heart. The blood-pressure, however, is naturally not governed solely by the heart and cardiac innervation, but by the resistance which the cerebral capillary vessels offer by virtue of their vaso-constrictor nerves, thus bringing in the higher automatic vaso-motor centres of the cortex, which are subject also to psychical influences, and the reflex vaso-motor centres in the pons and medulla as well as the associations-tracts, the reflex vaso-motor centres of the spinal cord acting only coördinately or subordinately.

This field of study is too broad to be entered understandingly in the present state of our knowledge. That vaso-motor paresis constitutes an important feature of the prodromal period of diffuse cortical encephalitis, and probably of other organic disease, as well as in the neuritis of lead and arsenic, must, I think, be admitted. The theory that there is a disease which is essentially of the cerebral vaso-motor centres, involving, perhaps, the associations-tracts and the spinal reflex centres, is one in favor of which much can be said, especially where the symptoms appear rather abruptly in people in otherwise good health, without marked emotional or other exhaustive antecedents, and where the least mental or physical effort produces marked cerebral and spinal hyperæmia. How far vaso-dilator nerves act in such cases

is a matter of doubt, the transient or more or less persistent anæmia and hyperæmia of the brain and cord being explained by excess of functional activity or inhibition of function in the vaso-motor centres.

10. The neurasthenic condition, in exalting the direct and reflex excitability of the nervous system, naturally intensifies the usual causes and conditions of insomnia, the unusual sources of insomnia in neurasthenia, in my experience, being astigmatism and hallucinations of sight or hearing. The eye-strain from astigmatism is often in health unnoticed, when in states of debility it produces headache, dizziness, spasmodic muscular action, or wakefulness.

An hallucination of sight occurring a single time is not uncommon in people in reasonably good health. Frequently repeated, such hallucinations are less rare than is supposed without any indications of mental or other disease. Occasionally, like flashes of light, they are precursors of headache. I have observed frequent hallucinations of hearing only once, independent of insanity.

If of distressing nature hallucinations of sight and hearing may be a fruitful source of insomnia. They occur beyond the power of the will of the individual to call them up, although sometimes able, under some conditions, to cause them to disappear. The hallucinations of sight constitute new arrangements of mental impressions which can be more or less clearly recollected, or they form combinations which seem entirely new. Once I have found two sisters subject to them, and once two sisters, a cousin, and a common grandmother—curiously enough, the different members of the families not knowing each other's peculiarities, which, however, were quite different in kind, until I began my investigations. They had thought them uncanny, and had concealed them.

11. The neuropathic temperament, usually by inherited predisposition, but which may be acquired. In its pronounced form it is closely allied to the well-marked functional diseases of the nervous system, and, at the critical periods of life, may readily develop into them. It is congenital, or due to early interference with the normal development of the brain, to faulty training, and to bad habits of living. It shows itself in infancy and childhood by irregular or disturbed sleep, irritability, apprehension, strange ideas, great sensitiveness to external impressions, disagreeable dreams and visions, romancing, intense

feeling, periodic headache, muscular twitchings. There are often excessive shyness, introspection and self-consciousness, or extreme self-assertion and conceit. The imitative and imaginative faculties may be quick, the emotions strong, the affections intense or almost absent. The natural feelings easily become disturbed and perverted. The passions are unduly a force in the character, which is commonly said to lack will-power. Self-discipline is a mighty task, and self-control is acquired only with great difficulty. The memory is now and then phenomenal. There is a ready reaction to external circumstances, even to the weather, by which the individuals become easily a little exhilarated or somewhat depressed. They are apt to be self-absorbed, and may be suspicious, or morbidly conscientious. Slight physical ailments, hardly noticed or rapidly recovered from in sound constitutions, leave on them a long or lasting impression. They readily become neurasthenic, hypochondriacal, or nervous invalids, so called, and they break rules or disregard established customs with less cause or provocation than other persons. They lack stability, or have in special directions narrowed limitations of intellectual energy, in quality or quantity. To the nervous temperament there may belong social and intellectual gifts and graces, originality, intensity, poetry, art, philanthropy. Adjusting skill, the ability to adjust their organism to their environment, to use Herbert Spencer's phrase, is often lacking.

Many of these people have the marks of Morel's *dégénérés* with few compensations; many illustrate that marvellous law of compensation in nature for defect in one direction by accomplishment in another. Often not only they, but generations before them, have been crammed mentally and emotionally but starved in other ways, their very abstemiousness producing a tough asceticism, requiring decades before the final but sure physical deterioration; or there may be a gouty diathesis and the two somewhat opposite temperaments, or perhaps constitutions, may be combined by intermarriage. They fail to store up nervous energy; they are unable to inhibit the free or excessive or explosive discharge of it in response to inconsequent excitations, and they are constantly in the position of having expended more than their reserve nervous force. From the prominence in adult life of the unstable emotional centres, which predominate in childhood, they are most difficult to treat. Most of them are insomniacs, more or less, to quote one of the most gifted and graceful of them. We can

no more relieve them of all their symptoms than we can add a cubit to their stature. But there are none in the community for whom we can do more, as there are none to whom alluring promises, including mind-cure, faith-cure, hypnotism, etc., are more seductive. "My physician stands between me and the devil," was the expression of one of them.

12. Wakefulness is one of the most difficult symptoms to treat in the various stages of many forms of mental disease, and is doubtless only an early symptom in many cases where it had been regarded as a cause. In hysteria, hypochondria, and organic diseases of the spinal cord and brain, including hemorrhage, embolism and thrombosis, insomnia often taxes our utmost resources.

13. As a form of insanity—that is, as an interchangeable psychoneurosis in families predisposed to mental disease—insomnia is not very uncommon, especially among Morel's *dégénérés*. In such case it is persistent for months or years, is attended with great mental and physical exhaustion from slight effort, and is most intractable to treatment. It usually ends in more or less permanent mental enfeeblement with impaired will-power and diminished self-control, perhaps without, but generally with, other psychical symptoms.[1]

In estimating the value of insomnia as a symptom—that is, in establishing a prognosis—it is chiefly in neurotic patients that we have great concern; and even in them it seems to me well always to bear in mind the distinction which Meynert draws between the large number *Zur Krankheit Berufenen* and the much smaller number of those *Zur Krankheit Erwählten.*

The one organ, excluding organic disease, upon the functional health of which I place the first dependence in prognosis, is the stomach.

[1] If this classification is unsatisfactory, the latest attempted on a scientific basis does not better meet the case, stated by Germain-Sée as follows:

1. L'insomnie douloureuse.
2. L'insomnie digestive.
3. L'insomnie cardiaque et dyspneique.
4. L'insomnie cérébro-spinale, neurosique, comprenant les lesions de l'encéphale, la paralysie générale, les manies aiguës et chroniques, l'hystérie, l'hypochondrie.
5. Les insomnies psychiques (émotives, passionelles et sensorielles).
6. L'insomnie de fatigue cérébrale et physique.
7. L'insomnie génito-urinaire.
8. L'insomnie fébrile, infectieuse, autotoxique.
9. L'insomnie toxique (café, thé, alcoöl).

There are certain obvious indications in the treatment of insomnia, to amend bad habits, which may mean the errors of a lifetime; to relieve painful impressions through any of the external senses; to remove depressing mental influences; to cease overwork; to interrogate every function and organ of the body; to correct constipation, faults of digestion, whether gastric or intestinal, oxaluria, etc; to forbid unsuitable articles of food and drink; to meet the indications of toxic influences in acute and chronic disease, from the abuse of tea, coffee, tobacco, alcohol, opiates, chloral, bromides, cocaine, or in poisoning from lead, arsenic, and other sources; to stop excesses and build up conditions of exhaustion and anæmia; to control as far as possible the vascular disturbances in hepatic, renal, and cardiac diseases, using leeches as needed in venous engorgement or congested conditions.

Further than this and treating symptoms, our measures must be largely restorative—so little do we know of sleep and sleeplessness or understand the pathology of the nervous system and its cells with their molecules estimated to contain each nearly a thousand atoms grouped together in small and large clusters, which are again united into larger more or less complex groups.

The amount of sleep needed either in health or disease and the injury from loss of sleep, vary so widely in different persons that naturally there can be no fixed rule for therapeutic interference. Generally speaking, the temptation is to resort to medical treatment too readily, and to be too much afraid of a few sleepless nights. In some individuals and in some families, a wakeful week, with little sleep each night, is not a matter for concern, as compared with acquiring the habit of depending upon narcotics; in others the use of sedatives, even for a long time, is attended with good results.

The hygiene of sleep includes the most minute attention to the whole environment of the patient, position, change of room or of locality, temperature of the body and of the air, diet, digestion, secretions, excretions, fresh air, soothing sounds or manipulations, rest and exercise accurately adjusted to the needs of the system, presence or absence of noise, relief from sources of irritation. When certain areas of the brain are overstimulated to exhaustion with wakefulness, the exercise of other parts, diverting the attention by pleasant conversation or fixing it upon a new subject, a short walk in the open air,

gymnastics, an agreeable novel, a light supper, koumyss, matzoon, malt or cautiously used wine, beer, ale, porter, spirits, are expedients which are useful some to one and others to another wakeful person. Change of climate, scene, and association, travelling, or, better, camp-life, especially in the mountains, a sea-voyage, or a wisely selected hydropathic establishment will often avail. Mountain-air has seemed to me best where there is increased vascular tension or diminished vasomotor control, especially in those cases where sea-bathing, tonics, and alcohol disagree. The sea-level and a humid atmosphere, in my experience, have been useful for the anæmic, the exhausted, and those with cardiac disease. Often the most suitable climate is best determined by trying one after another. We have the authority of Marcus Aurelius for the statement that Æsculapius prescribed horseback exercise. The outside of a horse has been said to be the best thing for the inside of a man. Under any and all circumstances I endeavor to get such patients into the open air, even when they are feeble, and into the air of the woods, or sea, or the country, if possible.

The application of cold to the head, heat to the feet or abdomen, of galvanism, faradism, dry cupping, the Paquelin cautery, Chapman's ice-bags to the spine, or prolonged hot baths, with or without cold affusion to the head, may be efficacious. Massage and the wet-pack are of the greatest value, and the treatment so properly identified with Dr. Weir Mitchell's name has made the only wise and efficient management possible for a large class, although, like all powerful remedies, capable of harm with unsuitable cases or in unskilled hands.

In neurasthenia and in conditions of unstable equilibrium, nervous or psychical, particularly where alcohol and most medicines fail or have some disagreeable after-effect, personal influence may outweigh all else; a teaspoonful of water taken with confidence becomes a hypnotic, and whatever develops or creates self-discipline and self-control in the patient is a remedy of no mean value. As Ruskin says, too, people think by infection, catching a passion like a cold. For these reasons the grotto of Lourdes, faith-cure, mind-cure, Christian science, spiritualism, hypnotism, have their devotees, and charlatanry may succeed where medical skill and experience fail. The physician's reputation or confidence in him from experience also go to make up no small part of the personal equation, and the so-called moral treatment

so long used with the insane has been, especially in the hands of masters like Bell and Ray, an immense power.

I am not prepared to say that hypnotic suggestion cannot be used to advantage in some cases of insomnia. The evidence thus far inclines me to the opinion of Germain-Sée and Gilles de la Tourette, that the French Ministry of War is right in interdicting its use by the army surgeons on the soldiers, on the ground of its tendency to do harm. To many in the community there may be no corresponding or compensating harm, to others the evil is great, from such a degree and kind of suspension of the higher cerebral activities—from losing their grip, so to speak—and one cannot fairly estimate its general effect on the community.

It is better, especially for people of the neuropathic temperament, to overdo and at times to suffer some sleeplessness in consequence than to sink into deeper and deeper inertia while striving to avoid every effort which brings discomfort with it, or after it, just as it is not always best to shut out every sound, even if disturbing to sleep. Many do best to bear a certain amount of wakefulness, as others must suffer pain, when our help avails much in giving strength and courage.

Stimulating tonics, phosphoric acid, phosphates, hypophosphites, quinine, strychnine, and iron may be serviceable, especially in small quantities, but certain patients exhibit a marked intolerance of medicines and alcohol.

When shall we use hypnotics? In acute disease, particularly fevers, sleep is often a necessity, reducing the activity of the heart, removing more waste, and quieting the general excitability of the nervous system. In chronic disease there is frequently the same temporary need. In incurable disorders with pain and discomfort, in the restlessness of senility, hypnotics and narcotics—and used freely in the last years of life—are almost the chief justification of our service. Often in mental disease they are, for a time, all but indispensable. In some neurotic people their occasional use can hardly be avoided. In acute nervous and mental disturbance from profound shock, full and continued doses of narcotics may dispel most threatening symptoms. The individual must, of course, be taken into consideration. Many can be depended upon to use hypnotics only as directed by their physician; others can no more be trusted with them than certain persons with alcohol. Something of slight intrinsic hypnotic value may be

intensified by its mental effect, and I am sometimes deliberately asked for a prescription upon which to build a mind-cure. It is often imperative to prescribe a hypnotic, where it is best that a decided hypnotic effect should not be got. For this purpose a somewhat unpleasant drug is better than an altogether agreeable one. The prescription which I use consists of a few minims of paraldehyde in a drachm of chloroform water. This can be repeated in the night several times, and be continued without harm.

In some conditions, even with acute maniacal symptoms, it is better to let the patient lie awake almost absolutely for two or three nights than give the amount of narcotics necessary to produce sleep.

The multiplication of sleep-producing drugs has its advantages in trained, skilful hands to meet idiosyncrasies of temperament and varying conditions of disease. But the immense production and widespread use of them in the last few years must have done incalculable harm.

Very many people—highly educated as well as ignorant—firmly believe that if there is not a specific for their malady the reason is that it has not been brought to light; and they not only demand repeated trials of useless remedies but assiduously follow enticing promises in regard to those that are even injurious. When our advice does not meet their fancies in that regard they continue their search for what they crave.

The greater one's experience, the more is he satisfied how little he can do as compared with what the community insists upon expecting, although more and more convinced of the great value of that which can be done, of the harm of overdoing, of the need of minute study in each case, and of the vagueness of general rules of treatment. The chief indications in insomnia are: To give hypnotic drugs rarely and only in cases when other measures have failed; to use, as far as is possible, the small dose, repeating as needed, so as to avoid the over-action or the cumulative effect of large doses; to be satisfied with the least amount of sleep that is safe if produced by medicine; to avoid drugs as a rule, except for euthanasia, when the mental condition is not such that all the after-effects can be noted; to bear in mind the fact that hypnotics given to produce sleep may increase wakefulness; to be on guard for unpleasant or toxic results when any drug is given in sufficient dose to produce prolonged and profound sleep; to give the large dose when it is indicated; not to expect the same action, or

tolerance, of new medicines in sensitive private patients as in hospital practice.

Of hypnotics, there are probably none that can be continuously used for a long time in sufficient doses to control troublesome insomnia without occasional dangerous symptoms or unpleasant after-effects, although I have no doubt that many of the accidents reported as due to them are rather coincident with them than consequent upon them. Most cases, however, are not intractable to treatment, and many respond readily to domestic remedies, like skullcap, thoroughwort, sage-tea, glycerin, or hot punch, without the physician being called, or perhaps their restlessness has been controlled by hyoscyamus, valerian, cannabis Indica, camphor.

The advantages of spirits, wine, beer, ale, porter (including some of the so-called malt extracts), koumyss, matzoon, are well known in old age, conditions of exhaustion, fevers, and generally where a cardiac depressant must be avoided, especially if supplemented by vaso-motor stimulants, like ergot, digitalis, strychnia, coca, cocaine. A glass of champagne often acts like magic in the sleeplessness of a dilated heart. A few bottles of beer at bedtime may control persistent and obstinate sleeplessness which has yielded to nothing else. But the habitual necessity of alcohol to produce sleep in brain-workers indicates the nearness of the danger line. The disadvantages of alcohol are well known and readily guarded against.

The preparations of opium are indispensable in many cases—with pain, in old age, in many forms of heart disease. I have not found the common caution against opium in pulmonary and renal disease valid, and probably no other drug can be made so happily to meet the indications of so many conditions, except for the grave danger of the morphine-habit. Krafft-Ebing names it justly a tonic to the asthenic brain. In euthanasia, its value is shared with only ether and chloroform.

The bromides diminish the reflex excitability of the medulla, and are vaso-motor excitants. Their best therapeutic effect, always avoiding bromidism, is got, as hypnotics, from divided doses given through the day or evening, whether in large quantity or small. Alone or combined with other medicines they have a wide usefulness. The debilitating influence from them may in some cases be, partly at least, met with iron or bark. By virtue of obstructing the heart's action,

and producing a certain degree of muscular depression, they are not without objections for continued use, or, in some cases, occasionally.

The great need of an unobjectionable hypnotic led to constant laboratory research, and the final discovery by Liebreich of *chloral-hydrate*,[1] Eickholt's *schlafmittel* κατ' ἐξοχήν, the most powerful of pure hypnotics, having also some influence on pain. In sufficient dose it rarely fails. Alone or combined with bromides and morphine, it used to be, until the introduction of paraldehyde, the common last resort when other narcotics had failed. Its depressing effect on the heart, respiration, and vaso-motor centres, which, perhaps, includes its toxic or irritant action on the kidneys, and the danger of the chloral-habit, with persistent wakefulness and mental enfeeblement, constitute the risks in its use.

Croton chloral, the butyl-chloral-hydrate, or, chemically, the chlorated aldehyde of crotonic acid, although not a cardiac depressant, is, in all other ways, so inferior to chloral hydrate, beside being not readily soluble in water, that it is rarely used except with trigeminal neuralgia, in which it is less efficacious than other drugs.

A less depressing hypnotic being sought for, the fat series came next, paraldehyde, amyl-hydrate, urethan, hypnone.

Paraldehyde,[2] a polymeride of ethylic aldehyde—long thought second only to chloral hydrate in power as a hypnotic—has much less depressing action on the circulation and respiration. It is especially valuable in conditions of mental excitement, where the duration of sleep may be prolonged by adding morphine. The physiological action is first on the cord, and then on the medulla. Toxic doses (10 grammes or more) produce a sinking of blood-pressure and slowing of the heart-beat; doses of 6 to 8 grammes having been observed to give rise to nausea, headache, confusion, vertigo, and weak pulse. In five cases Berger found material reduction in the quantity of urine. In safe doses it is not anæsthetic or analgesic. To get a definite hypnotic effect, the dose must be increased. Its acrid taste, and the disagree-

[1] An abstract of recent articles on the new hypnotics was intended as an appendix to this paper, but the amount of material, over a hundred foolscap pages, was too large to justify its space; and its appropriateness here, whether in full or condensed, has seemed to me at least doubtful.

[2] Albertoni, Berger, Butler, Cervello, Cervello and Valenti, Clouston, Fronmüller Fröhner, Henocque, Hodgson, Kenniston, Krafft-Ebing, Liebreich, Peretti, Quinquarc, Rank, Rothe, Strahan, Tchepetow, v. Noorden.

able persistent odor from the breath are objections to its use, and even when largely diluted it is often objected to, on the ground of its being a gastro-intestinal irritant.

The temptation to its habitual use is less than in alcohol, morphine, cocaine, and chloral; the symptoms produced being tremor, confusion, impaired memory, diminished intelligence, etc.

Dose, two to four grammes, which, if necessary, may be gradually increased to eight, and repeated once or twice in the night.

Amyl-hydrate,[1] a tertiary amylic alcohol, an oily colorless liquid, appears to stand in hypnotic power below chloral hydrate, and above or below paraldehyde, according to different observers, but with even less depressing action on the heart than the latter. It may be used in about the same dose as paraldehyde, being not so soluble (1 to 19 water, freely in alcohol), and with a much less disagreeable taste and odor. Its physiological action is directly on the cerebrum, and later on the medulla. The toxic effects from it, of headache, nausea, weak pulse, are less marked than in paraldehyde, and it is less a gastro-intestinal irritant, although sufficiently so often to require its administration by rectum.

Urethan[2] (ethylic carbamate), a mild hypnotic, in doses of twenty grains, is at times useful, and may from its ready solubility be given hypodermically. It is without special depressing action on the heart, except in enfeebled persons. It has, like all rather mild remedies, proved in some cases uncertain in its effects, but is useful and ordinarily without disagreeable after-effects.

Hyoscyamin,[3] isomeric with atropine, in doses of $\frac{1}{2}$ mgm. of the sulphate, which may be given subcutaneously, controls moderate mental excitement and sleeplessness. In larger doses similar toxic effects to those of atropine are observed, sometimes with muscular prostration, which may reach an alarming degree. Of some value in hysteria with violence, its use is very limited.

[1] Buschan, Dietz, Égasse, Eskoff, Jolly, Griffith, Gürther, Lehmann, Liebreich, Mason, Mayer, Riegel, Saves, Scharschmidt, von Mering, von Mering and Thierfelder, Willis, Wurtz.

[2] Adam, Demme, Griffith, Sansom.

[3] Clin, Égasse, Geiger, Hesse, Horn, Ladenburg, Lemoine, Reichard and Duquesnel, Runge, Thompson, West, Will.

Hyoscin,[1] also an alkaloid of hyoscyamus, is used as iodohydrate, chlorhydrate, and, preferably, hydrobromate, also hypodermically, in doses of $\frac{1}{5}$ to $\frac{1}{2}$ or even 1 mgm. It has a limited usefulness, especially in maniacal excitement, violent hysteria, delirium tremens, insomnia with agitation, in the insane and aged. The dose needs to be increased somewhat rapidly for prolonged use, and a slight excess in the quantity given may produce unpleasant toxic effects, which may even come from $\frac{1}{2}$ mgm. In a certain proportion of cases not only is there no quieting effect from the drug, but the restlessness is increased.

Chloralamide,[2] or more properly chloral-formamide, produced by adding anhydrous chloral to formamide, is decomposed again in aqueous solution (1 to 9) if above a temperature of 60° C., or in crystals, if above 115° C. It is also decomposed by alkalies and alkaline carbonates. It is, therefore, well to give it in slightly acid solution with spirit or as an elixir, thereby sufficiently disguising its bitter taste. It is claimed that it possesses all the advantages of chloral hydrate, and, on account of the stimulating properties of the formamide, without its depressing action on the heart, a fact verified by the sphygmograph, the comparative dose being as 3 to 2. The danger from the larger dose, over 30 or 40 grains, is less than in chloral. Disagreeable after-effects are much less common. Some observers value chloralamide most highly, while others regard it as in no way deserving especial praise. In my experience, with only the small dose, it has done well. If it shall be proved, after full trial, to be safer than chloral, and more certain than the other pure hypnotics, as it is claimed to be, its cheapness is in its favor for general use.

Sulfonal,[3] diethylsulphondimethylmethan, is a less powerful hypnotic than chloral, with even less influence on pain, but without so much, although with slight, depressing action on the heart. From its difficult solubility, the physiological action is slowly produced; a single dose

[1] Buddee, Edlefson and Illing, Égasse, Erb, Fischer, Githeus, Gley and Rondeau, Hahn, Kny, Konrád, Krauss, Ladenburg, Magnan and Lefort, Mairet and Combemale, Malfilâtre and Lemoine, Mitchell, Peterson and Langdon, Pitcairn, Reichard, Salgó, Thompson, Weber, Wentherell, West.

[2] Alt, Aulde, Cope, Égasse, Hagemann and Strauss, Hagen and Hüfler, Kalacz, Kny, Langgaard, Leech, Letton, Mehring, Norman, Osler, Paterson, Peiper, Rabow, Reichmann, Robinson, Schaffer, Setlow, Strahan, White, Wurtz.

[3] Bornemann, Cramer, Fischer, Fränkel, Funajoli, Raimondi, Griffith, Henocque, Kast, Kisch, Knoblauch, Laudan, Liebreich, Müller, Otto, Rabbas, Rosen, Rosenbach, Salgó, Schmey, Schwalbe, Steiner.

may be efficient for two successive nights, and there is risk of the cumulative effect if the dose is too often repeated. It may be given in compressed tablets, or wafers, or dissolved in spirit. In doses of 30 grains or less it is, in the main, safe, although I have seen 20 grains given each night for several weeks in a girl of sixteen produce stupor, and 20 grains, three times a day continuously, to a strong male adult, cause ataxia, tremor, and mental apathy. It has been found not always certain, and, in over-action, with an after-effect of somnolence, fatigue, depression, lack of appetite, which, from an overdose, may amount to semi-coma and cardiac depression. Given in doses of 5 grains, and repeated, if necessary, two or three times, or of 20 grains, and perhaps repeated, it has proved, in my hands, an hypnotic of great value in producing sleep which is refreshing and resembling the natural. When care has been taken not to give it too often or in too large doses, I have never seen any untoward results from it. In the case of a rather delicate person, 150 grains produced sleep with stupor for ninety-six hours, but no other unpleasant symptoms, except some lassitude and a sense of discomfort in the head. It is the most important and widely useful of the new hypnotics.

Hypnone[1] (acetophenone, phenyl-methyl acetone), one of the aromatic series of acetones, although praised by Dujardin-Beaumetz, has proved in the hands of other investigators of little value as an hypnotic, of unpleasant taste, and depressing to the respiratory and cardiac centres.

Ural[2] (chloral-urethan) is less potent than chloral, and more so than urethan. It is very bitter, soluble in alcohol, and but little so in water. In dose of more than two grammes there may be transient headache and fatigue.

Somnal[3] (ethyl-chloral-urethan) contains four atoms of hydrogen and two of carbon more than chloral-urethan (ural). It appears to have no especial advantage over chloralamid, except that it is more soluble. It has somewhat greater hypnotic power than ural. Its composition is not regarded as certain or its action sure.

[1] Bardet, Dujardin-Beaumetz, Friedel, Hirt, Kamenski, Kny, Kraepelin, Krauss, Laborde, Mairet and Combemale, Norman, Rottenbiller, Seifert.

[2] Albertoni, Bischoff, Compari, Poppi.

[3] Égasse, Paul, Radlauer, Robinson.

Acetal[1] (diaethylacetal) is an acrid hypnotic without advantages to compensate for its being a gastro-intestinal irritant.

Methylal,[2] an acetal derived from formaldehyde and methylalcohol, a volatile liquid freely soluble in water, of fragrant and aromatic odor, almost tasteless, may be inhaled or given by mouth, and hypodermically. It has the high authority of Krafft-Ebing as being a safe, certain hypnotic, useful in some forms of insanity with excitement, and especially in delirium tremens, for which he considers it the best hypnotic known. He uses 0.1 gramme with 1 gramme water, hypodermically, repeating every two hours if necessary. The dose internally is ♏ 30 to ʒij. The depressing effect on the circulation is slight, if any, and he has not observed disagreeable after-effects. It is contraindicated in conditions of active cerebral hyperæmia. Krafft-Ebing is in doubt how to explain the efficacy of the extremely small dose used by him, unless it acts indirectly rather than as a direct hypnotic in producing sleep; Lemoine gives 2 grammes subcutaneously.

Phenacetin,[3] para-acet-phenetidin, almost insoluble in water, soluble in alcohol, almost tasteless, may be given like sulfonal, in wafers and compressed tablets, as a powder or with brandy. As an antipyretic and antineuralgic it is not as potent as antipyrin and antifebrin, but it is much less a cardiac depressant. In the insomnia of overwork, of nervous irritation, in febrile states, or from headache, it is an hypnotic of great value, in doses of five or ten grains, repeated if necessary. In sleeplessness from intense neuralgia, less than a gramme repeated two or three times, as needed, is not likely to be effectual, the fact having been first ascertained that there is no intolerance of the drug.

The possible anilin derivatives of which phenacetin (para-aethoxy-acetanilid), acetanilid and methacetin (paramethoxy-acetanilid) have thus far alone been used in medicine, together with numerous known compounds of not ascertained value, indicate the boundless possibilities for the future in this direction.

Acetal, ural, hypnone, and somnal do not seem to me to have sufficient therapeutic value to justify their existence. Hyoscin and hyoscyamin have a limited range. Of the other hypnotics we must

[1] Berger, Hiller, von Mering.

[2] Haldjès and Boubila, Krafft-Ebing, Lemoine, Mairet and Combemale, Matrokhin, Personali, Popoff, Richardson.

[3] Ayers, Heinz, Hensner, Hinsberg, Rumpff.

trust somewhat to experience, in ascertaining which is least likely to disagree with a given patient; and to one already discouraged by long illness an unsuccessful trial of a new remedy may be so unsuccessful as to be disastrous. Personally, I use them sparingly, and as indicated. There are cases when very large doses of powerful hypnotics must be used to produce sleep, or else alcohol freely. On the other hand, most patients sleep better than they think, and many sleep enough who believe that they sleep scarcely at all.

DISCUSSION.

DR. H. M. LYMAN: It seems to me that much of the difficulty that we encounter in studying the physiology of sleep grows out of the fact that the brain is not a simple organ, but is a cluster composed of a great number of organs; and the differences that we observe in varieties of insomnia grow out of the fact that each individual case is different from every other. In one case it is one organ, or set of organs, that is over-exhausted or deficient in its action; in another, it may be another. So that it is necessary to study the physiology of each individual case of sleep and insomnia in order to interpret the physiology of the subject.

But there is no time to discuss this. I simply wish to ask Dr. Mitchell, with regard to the rigidity of the limbs occurring in what I think he calls the post-dormal state, whether it might not be referred to the anæmic condition—perhaps, to the unusual persistence of anæmia during that state. I have been inclined to refer to that cause the condition of which the Doctor speaks, and to regard it as one of the consequences of a starved condition of the nervo-muscular organs.

In a large class of cases of insomnia we have to deal with the rheumatic diathesis. I have found the use of sulphur very beneficial in such cases, both in the way of employing mineral waters containing sulphur, and lately in that combination to which Garrod, of London, has recently called attention in *The Lancet*. I have found no hypnotic as beneficial in that class of cases.

DR. G. L. PEABODY: As one important practical point dwelt on by the reader of the last paper, it is well to emphasize the fact that disturbance of the digestion and assimilation may often be a cause of protracted insomnia, without there being any of the ordinary manifestations of indigestion. I think Dr. Folsom dwelt upon that, and, so far as I know, he is almost the only writer who has dwelt upon the fact that disorders of digestion, without producing any of the ordinary symptoms, may, at the same time, be the cause of

protracted and very obstinate insomnia. A case of this kind came under my observation a few years ago in the shape of a young man who was a student at a German university, who had been treated for insomnia nearly a year by two professors in the university whose names would be familiar to you all. One—the professor of nervous diseases—told him that he was suffering from cerebral hyperæmia; that he must dine earlier in the day, drink beer at night, even in the quantities recommended by Dr. Folsom, and that his symptoms would abate. He followed the prescription, and the symptoms did not abate. He went away and took a protracted vacation, spent several months in the open air, and got well. He returned to his studies, and the symptoms returned. He then went to the professor of medicine in the same university, who told him that his brain was anæmic, and that he ought to take iron and certain other remedies. He resorted to chloral, and became a confirmed chloral-taker; and it was only when he came home and put himself under the care of his family physician, and took rhubarb and soda, that he got entirely well.

Dr. Wharton Sinkler: Dr. Mitchell has referred to the disorders of sleep in children. I am satisfied that the loss of sleep—this disturbance of their sleep, dreams, nightmares, and restlessness—arises in many instances from hypertrophied tonsils.

Dr. M. Allen Starr: It seems to me that this discussion would be incomplete if no mention were made of the use of sulfonal in the treatment of disorders of sleep. It has seemed to me so far superior to all other remedies that I cannot help mentioning its use, largely for the purpose of calling attention to its great efficacy in the disorders of sleep that occur as an early symptom of mental disorders. It has proven to be a great deal of service in my experience. If it is given subsequently to placing in the stomach some article of food it has a more immediate and lasting effect than if it is given on an empty stomach. When given within twenty minutes of a glass of milk or some simple nutriment it has proven efficacious in doses of twenty grains.

Dr. S. Weir Mitchell: I give my decided assent to the points made by my colleague. The mass of people troubled with insomnia belong to the neurasthenic class which he described. Among these are people whose attention has been called to the question of sleep, whom we might with reason call insomnomaniacs. They watch the process of going to sleep with such attention that it is inhibitory of sleep. I think Dr. Folsom has met with enough of this kind of people to know they are among the most difficult cases. Very often you have so to impress them with some drug for a number of nights as to teach them that long slumber is possible. It is also frequently necessary to do this in some shape with persons who do not get well of their other troubles—gastric embarrassments, etc.—unless you give them sleep first. That seems to be the key to success. Get them to sleep first, and everything else follows.

As concerns the class to which you want to insure full sleep in quantity I am in the habit of giving bromide, not at night, but in the daytime, twenty or thirty grains three times a day for the first week and then diminish it by degrees, never allowing the patients to handle their own drugs or know what they

are taking, if it can be avoided, keeping the whole business in my own hands or that of the nurse. I think the best plan is to give the large dose first and then to lessen it, instead of beginning with a small dose and increasing it.

As to the question asked by Dr. Lyman about rigidity occurring from anæmia, I say that I have seen but two cases of the profound rigidity to which I have alluded, and in neither was the patient anæmic; the conditions presented were apparently those of absolute health.

A STUDY OF THE ANÆSTHESIAS OF HYSTERIA.

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THE object of my paper is to report the results of a study of the anæsthesias of hysteria. This is a subject upon which a great deal has been written; but, as those familiar with hysteria will admit, there still remain many questions to be settled, and their importance, I think, need not be urged. Objective symptoms are always very greatly desired in medico-legal cases as well as for guidance in diagnosis and treatment, and anæsthesia in some of its forms has the value of an objective symptom. The anæsthesias found in hysteria may, it is true, be sometimes caused by organic disease, but this is not the case often, nor can organic disease ever present the same association of symptoms as those found in the profoundly marked cases of hysteria.

I shall not go into any extended history of the study of hysterical anæsthesias. The credit of initiating and carrying out investigations of the special sense disorders belongs to Charcot, Galezowski, Landolt, Badal and Binet, Pichon, and Charcot's pupils, Turette, Guinon, and others. Special articles have appeared in Germany by Oppenheim, Thomsen, Moravcsik, Babinsky and Schiele. Among English writers, Thorburn, H. Griffith, Beevor, Bastian, and Buzzard; and in this country, Putnam, Walton, W. O. Moore, Mitchell and de Schweinitz have made important contributions.

My studies were made upon thirteen cases of hysteria, in men and women, of traumatic and non-traumatic origin. Most of them were cases under the observation for a long time of myself and other neurologists and in all there was perfect agreement as to diagnosis, a diagnosis justified in several cases by the subsequent complete recovery of the patients.

The histories of my cases illustrate very well the four points in regard to hysteria which modern researches have tended to bring out. These are: 1. The comparative frequency of the disease in men. 2. The common characteristics of the disease, whether caused by shock, trauma, local irritations, or general depressing influences. 3. The presence of some of the objective symptoms or stigmata of the disease in all cases. 4. The combination of true hysteria with organic disease.

As I have stated, the class of symptoms which I particularly investigated was the anæsthesias. The hyperæsthesias, pains, paralyses, tremors, spasms, crises, etc., are referred to incidentally and not in detail. Nor have I time or space to give full reports of each case. I shall only present enough to justify my diagnosis.

The points especially noted were the cutaneous sensations of touch, temperature and pain, their disorders and the distribution of them, the muscular and articular sensations, the vision, hearing, taste, smell, and the reflexes.

The cutaneous, the muscular, and the articular sensations were examined in the usual way. The deep sensibility was tested by thrusting needles into the periosteum and joints. The vision was tested for acuity, visual field, and color-sense. The visual fields were tested with Emerson's perimeter. The fundus was examined by myself or others. Hearing was tested for acuity both by aërial and bone conduction. It was also tested for high notes by means of Galton's whistle and for low notes by means of a bass viol string which gave a note of sixty to seventy vibrations per second.

It will be seen that the persons whose histories I relate are not sufferers from the grand hysteria of La Salpêtrière, with cycles of convulsive seizures. The patients were generally quiet, much depressed mentally, discouraged by suffering, lacking vitality, hopefulness and volitional power. They were made ill by depressing influences, hard work and poor food, or by injuries or profound mental shocks. Seven were men, and they were among the most typical cases. Nearly all were of foreign birth or immediate descent, and generally of German, Polish, or Russian origin.

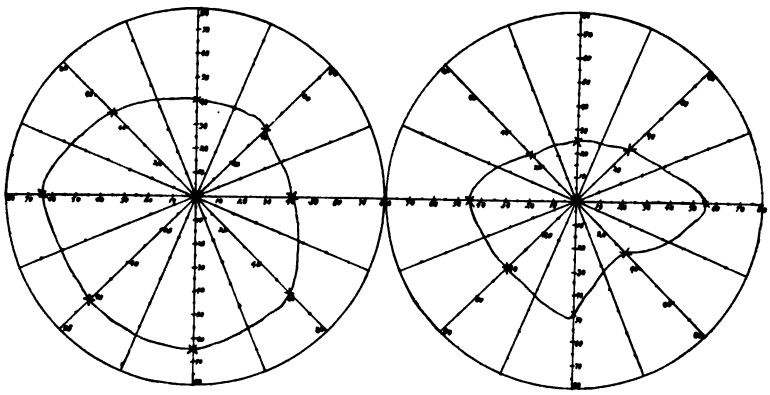
CASE I. *Hysteria with anæsthesia.*—Woman, aged twenty-four years, Hungarian, cloak-maker; childbirth, puerperal fever, abdominal and ovarian neuralgia, oöphorectomy. Symptoms date back three years to birth of a child and fever. Mental depression, apathy, general weakness, neuralgias, no paralysis.

Sensory symptoms: Has analgesia and tactile anæsthesia in patches over limbs. No thermo-anæsthesia or ataxia. Eyes: Vision good, visual field limited in both eyes, color-sense not tested. Hearing normal. Taste normal. Smell normal. Discharged unimproved and is now in about the same condition.

CASE II. *Hysterical hemianæsthesia.*—Moses S., married, tailor, German, aged fifty years. Family history negative. Personal history: Has been in good health until seven years ago, then had headaches and nasal trouble. Three years ago had attacks of transitory neuralgia of inframaxillary branch of trigeminus with aphasia. Then had and still has occasional diplopia, impaired vision. Attacks of a syncopal character lastly developed, and obliged him to give up work and enter the Montefiore Home, June 2, 1889.

Status præsens: A small man, prematurely old, quiet manner. Slight weakness of right arm and leg; no facial or ocular paralyses.

CHART I.



O. S. V. $= \frac{20}{200}$: with $+ \frac{1}{36} = \frac{20}{40}$.

O. D. V. $= \frac{20}{200}$: with $+ \frac{1}{36}. = \frac{20}{40}$.

Color perception perfect. Dacryocystitis.

Sensation—Cutaneous: Tactile and pain anæsthesia on right side. Temperature-sense preserved on examination in June; later (September) it disappeared. No loss of muscular or articular sensation; no ataxia. Mucous membrane of mouth hemianæsthetic; pharyngeal reflex not abolished on either side. Eye: Concentric limitation of visual field, more in right eye; no disturbance of color-sense. Fundus showed no special change; has a presbyopic eye. Ear: Some diminution of acuity of hearing in right ear, especially to bone conduction. Limitation of hearing to high and low notes in right ear. Taste, hemiageusia. Smell, hemianosmic. Knee-jerk lessened, especially upon the right side.

During the subsequent seven months the patient had attacks of the nature of spontaneous hypnotism. In one attack the Home physician, Dr. Ettinger, was suddenly called to patient, who was declared by the nurse to be dying. Pulse slow, strong, and full; temperature and color normal; pupils normal; pupillary reflexes preserved. State pronounced to be the hypnotic. Aroused by blowing into his face and irritating the conjunctiva with the pulp of one finger. A few passes made over the eyes sufficed to cause the patient's return to his former condition.

April, 1890: Patient still exhibits his hemi-paresis and anæsthesia, but is better of his hypnotic and neuralgic troubles, and is stronger.

CASE III. *Hysterical anæsthesia and vomiting.*—Israel S., married, tailor, German, aged thirty-seven years. Family history negative. Personal history: No illness until three years ago, when he began to suffer from headaches, left-sided pains, and attacks of vomiting.

Entered the Montefiore Home June 12, 1887. From that time to September, 1889, he presented about the same symptoms. He vomited food daily, generally ten to fifteen minutes after taking it, and without nausea. Despite continual vomiting he lost no flesh. Has no crises except of vomiting and pains in head; mental condition depressed. Forced feeding, medicinal treatment, lavage, did little good. Weight continued 108 to 110 pounds.

Sensation—Cutaneous: Complete bilateral analgesia, except in first branch of trigeminus, where it is less marked. Thermal sense not noted. Tactile anæsthesia over same area less complete. No muscular and no articular anæsthesia. No ataxia. Eyes: Vision normal; concentric limitation of visual field of both eyes. Reflexes: Exaggerated knee-jerk. At one time he had retention of urine.

CASE IV. *Hysterical paraplegia.*—Mrs. Mary D., aged twenty-eight years, widow, Hungary, seamstress. Family history negative. Personal history: Well up to five years ago; first child at twenty years, normal labor; second pregnancy at twenty-third year, during it lost her husband suddenly; labor at term was difficult, and was followed by much menorrhagia. Since that time has had three operations on the womb for laceration, etc. February, 1889, double oöphorectomy was performed. Since then she has been unable to walk; has attacks of crying with sensation of complete powerlessness; has less pain than before operation. After admission she had hysterical convulsions which could be controlled by ovarian pressure.

Status præsens: A dark sallow, anæmic-looking woman, muscles well developed, has incomplete paraplegia, and walks with great difficulty.

Sensation—Cutaneous: Analgesia over right hand, and in patches over arm, less in left arm. A zone of anæsthesia over abdomen between umbilicus and Poupart's ligament. Loss of tactile-sense is slight. No thermal or muscular anæsthesia. Ear: Limitation of auditory field in left ear to high notes. Bone conduction and low notes not tested. Eye: Visual acuity normal; concentric limitation of visual fields of both eyes. Taste normal. Smell normal. Patient improved under hydrotherapy, pack and half-bath daily, and faradic brush. She was discharged cured in November, 1889.

CASE V. *Hemianæsthesia, brachial monoplegia.*—Sarah C., aged twenty years, domestic, Galicia. Family history negative. Personal history: Good health until two years ago, then began to have attacks of vertigo, headache; would fall to the ground, sometimes with loss of consciousness. After some months she began to lose power in the left arm. A year ago entered Bellevue Hospital; here her seizures ceased under moral treatment, but after two months her monoplegia suddenly became complete.

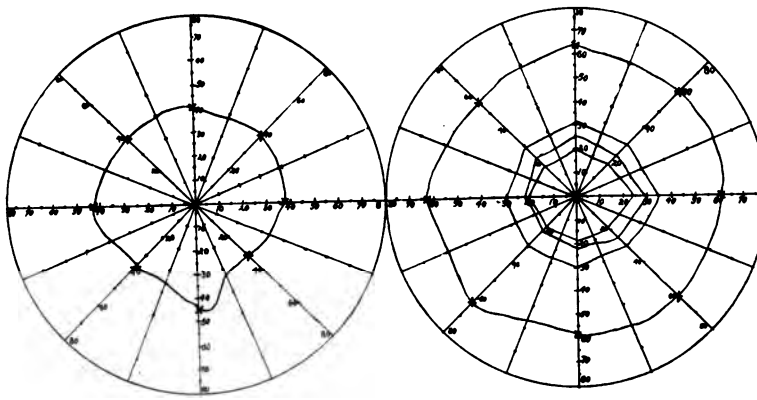
Status præsens: Looks old for her age, muscular and adipose tissue well developed. Height, five feet three inches; no disturbance of facial or ocular muscles. Pupils react normally. Complete flaccid paralysis of left arm; she cannot move arm, forearm, or fingers; no atrophy at all, as shown by measurements. Electrical reactions show slight quantitative diminution of sensibility and contractility to both currents.

Sensation—Cutaneous: She has cutaneous hemianæsthesia of the left side as far down as the knee. At one time it had included the whole of the left side. There is loss of tactile, pain, and temperature-sense. Muscular- and articular-sense still somewhat retained; she knows the position and movements of her arms, but not of her fingers. Eyes: Concentric limitation of visual field of right eye. Amblyopia of left eye. She can see light, and, to a certain extent, form, but cannot recognize colors or distinguish the nature of objects. No disturbance of color-sense in right eye. Mucous membrane of mouth anæsthetic. Pharyngeal reflex absent on anæsthetic side only. Ears: Some deafness in left ear; watch heard at three inches from left ear, at ten inches from right ear. Slight bone deafness. Marked deafness to high notes, as tested by Galton's whistle, and to low notes, tested by bass viol string. Smell, left hemianosmia. Taste, hemiageusic. Tendon reflexes lessened on affected side; organic reflexes normal. Patient is depressed mentally, but is very anxious to get well, and has no crises. Has been hypnotized several times with no benefit; is improving under hydrotherapy and electricity and strychnine hypodermically.

May 12. There is considerable return of motor power in the left arm; at first there was much tremor, but it is subsiding with the increase of strength. *Cutaneous sensations*—arm and face: Tactile sense is slightly diminished, especially for localizing, but very light contact is felt without delay. Pain-sense is still nearly abolished. Temperature-sense is entirely absent to both heat and cold. Pain is felt, however, in the periosteum when the bone is struck by a needle. Weight-sense is impaired, but articular sensations, as tested by posing and coördinating the limbs, are perfect. There is no ataxia. Vision: There is some dimness of vision in the right eye, but no loss of color-sense. In the left eye there is loss of color-sense, and almost complete loss of vision. She recognizes a light, and imperfectly a form, but cannot tell how many fingers are held up. With colored glass over this eye she has monocular diplopia on repeated tests. With colored glass over both eyes she has diplopia. With colored glass over both eyes she also insists that the image of the left eye is smaller—*i. e.*, micropsia. The visual field is contracted most in the left eye, where there is also loss of color-sense. In the right eye there

was a little change in the arrangement of the colors, but no inversion of formula, except that it was green, red, violet, instead of violet, green, red. Hearing to the watch in right ear at ten inches, left ear three inches. Deafness to tuning-fork in left ear when vibrating on the teeth or the mastoid. Deafness in left ear to high note and low notes.

CHART II.



O. S. V. $= \frac{5}{200}$: with $- \frac{1}{20} = \frac{20}{40} +$. Perioptic choroiditis atrophica.

O. D. V. $= \frac{6}{200}$: with $- \frac{1}{20} = \frac{20}{40} +$.

Color-sense absent in left eye. Field limited, with some changes in right eye, as shown by chart. The order of colors from within out was green, red, blue, violet, yellow.

CASE VI. *Hysterical paraplegia.*—Fanny H., widow, aged fifty-two years, Prussia, seamstress. Family not neurotic. Personal history: Three convulsive seizures at the beginning of menstruation; menorrhagia and various minor uterine troubles during her life; married twice, had one child and one miscarriage; was anæmic and overworked for some years prior to present trouble; always constipated and had some rectal trouble from operation to which she ascribes her present illness. This began in December, 1888, with severe lumbar and pelvic neuralgias. Tremor in arms developed rapidly, and, at the same time, weakness in the lower limbs.

Status præsens, September, 1889, nearly a year after the disease began: She has paraparesis more in right leg, and walks with difficulty. Right arm weaker than left. A fine tremor increased on voluntary movement, most marked in right arm but somewhat in left. Ocular muscles normal; no facial paralysis.

Sensation—Cutaneous: Patches of analgesia over feet and ankles. No tactile or thermic anæsthesia or ataxia. Knee-jerk somewhat exaggerated. Eyes: Vision good, slight temporal limitation of visual field in both eyes (65° in

each). Ears: Hearing good; no bone deafness or limitation of aural field. Taste normal. Smell normal. This patient improved steadily under treatment, and was discharged cured. The symptoms suggest multiple sclerosis, but the course of the disease disproves it. She was markedly neurotic and emotional, but had no crises.

CASE VII. *Hysterical paraplegia.*—Augusta K., widow, button-hole maker, German, aged forty years. Personal history: Since age of thirty-four has had occasionally attacks of rheumatism. Has had nine children. No nervous disorder. Lost the use of the right arm for two months and began to develop paraplegia and various forms of neuralgia, especially spinal.

Status præsens, two years later: Patient is in good health, but flabby and anæmic. Mentally depressed and melancholic. Paralysis so that she cannot walk except with support. No spasms or tremor. Muscles not atrophied except from disuse.

Sensation—Cutaneous: Bilateral analgesia and tactile anæsthesia over four extremities but not face; no thermo-anæsthesia or ataxia. Eyes: Vision good. Pharyngeal reflex normal. This patient continued for nearly two years paraplegic and suffering from neuralgias and headaches, occasionally vomiting and dysuria. She is now very much improved, and her anæsthesias have disappeared.

CASE VIII. *Hysterical incontinence and vomiting; hemianæsthesia.*—Fanny G., single, Russia, seamstress, aged twenty years. Family history negative. Personal history: Had no special illness until her fifteenth year. Then after exposure had dysuria and bloody urine. Suffered from this at intervals ever since. Came to the United States one and one-half years ago, and for two months was well; then suddenly developed cystitis and retention. Has had two vaginal operations and a suprapubic cystotomy done for relief of vesical pain, spasm, and cystitis, but with no success.

Status præsens, September, 1889: Patient is of small stature, somewhat thin. She remains in bed most of the time, and cries frequently on account of her pains. She is depressed and emotional, and tends, it is believed, to exaggerate greatly her sufferings. No paralyses or spasmodic symptoms. She has hysterical crises of an emotional character.

Sensation—Cutaneous: She has analgesia in patches over the extremities, more on the left side; no tactile or thermo-anæsthesia or ataxia. Eyes: Vision good; field much limited concentrically in both eyes and decidedly. Other special senses normal.

March, 1890, six months later: The patient's bladder ceases to trouble her, and she now has obstinate vomiting.

Sensation—Cutaneous: She has left hemianæsthesia to touch and pain, very slightly to temperature; pharyngeal reflex absent on the affected side. Eyes: Vision good, field limited, no disorder of color-sense. Smell normal. Taste normal. Hearing: Hyperæsthesia; no limitation of field to high notes.

CASE IX. *Hemiplegia from injury; hysterical anæsthesia.*—M. L., married, German, aged forty-seven years. Five years ago injured in right side of neck by an iron rod, causing, it is alleged, a perforating wound. Injury

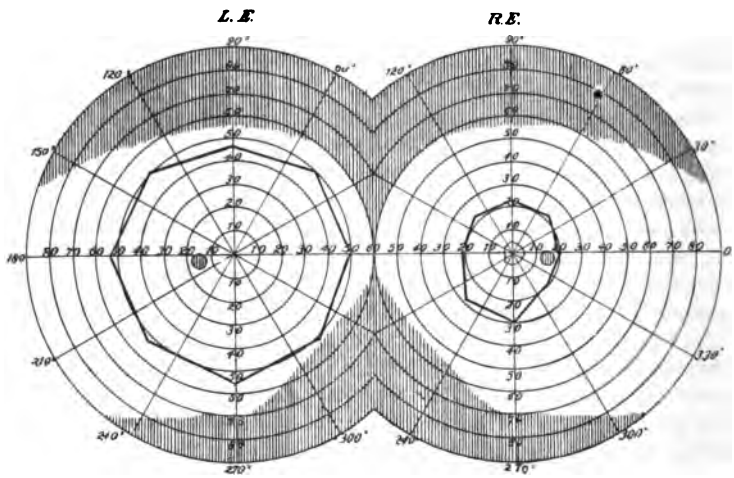
followed by right hemiplegia and right facial paralysis. Operated on by Dr. Robert Abbe for left tic douloureux with great temporary relief, the second branch of the fifth nerve being removed.

Status præsens, April, 1890: Patient is a well-nourished, healthy looking man, complaining only of neuralgia in the left inferior maxillary nerve. He has paresis and some rigidity of the right arm. Dynamometer: Right hand, 30; left, 45. He has apparent paresis of left leg, but tested on pedometer he pushed 140 pounds with the left leg and 100 pounds with the right leg. No facial or ocular paralysis. Tendon reflexes exaggerated on paretic side.

Sensation—Cutaneous: Anæsthesia of left side of face and right side of body and extremities. Left face: Loss of tactile and pain-sense. Right arm and hand: Diminution of tactile and pain-sense; retention of cold-sense and weight- and muscle-sense. Eyes: Vision good, but is color-blind; concentric limitation of visual field in both eyes; fundus normal. Taste: Left hemiageusia. Smell: Left hemianosmia.

CASE X. *Traumatic hysteria, hemiplegia, and hemianæsthesia.*—Adolph N., married, aged fifty years, Moravia, clerk. Family history negative. Personal

CHART III.



$V = \frac{15}{60}$. Inner circle shows limitation of visual field for colors.

history: A man of nervous temperament but good habits, and no previous nervous disorder. Was struck on the head by a dead electric light wire, knocked down, taken in a semi-conscious state to a hospital, where he was found to have right hemiplegia, except of face, eyes, and tongue, right-sided intention tremor, right hemianæsthesia, mental depression, no crises, knee-jerk lessened.

Sensation—Cutaneous; Right side anæsthesia most marked in extremities, well marked for pain and cold, less marked for tactile sense; no muscular or articular anæsthesia. Eyes: Decided limitation of visual field, especially in right eye; no loss of color-sense; Fundus normal. Hearing: Good to voice and watch in both ears, but complete bone deafness in right ear, and deafness to high notes and to low notes. Smell: Left hemianosmia almost complete. Taste: Left hemiageusia. Pharyngeal reflex absent.

CASE XI. *Hysterical paraplegia.*—Mrs. K., married, aged thirty-two years, United States. Neurotic family. Had incontinence of urine and migraine as a girl. Married at nineteen; child born at twenty; after this better for several years. At age of twenty-eight pains in back and legs, very severe, with weakness of lower limbs. Was treated gynecologically for a long time. Now has paraparesis and pains in legs; no bladder trouble, atrophy or anæsthesia; knee-jerk normal. Has insomnia, nervous crises, limitation of visual fields, but no color-change or auditory or other special sense troubles.

CASE XII. *Hysterical paraplegia, traumatic.*—William B., aged twenty-six years, single, United States. Injured by fall in August, 1885. In St. Luke's Hospital from October, 1885, to May, 1886, and had convulsive attacks there. Right hemiparesis, especially of leg; walks with difficulty. No atrophy, but exaggerated knee-jerk and ankle clonus. Slight analgesia, right leg and lower half of thigh and right forearm and hands. Thermo-anæsthesia over same area; contact sense good. No muscular anæsthesia. Concentric limitation of visual field; scotoma for red (due to tobacco?). Fundus shows a tobacco optic atrophy.[1] Auditory field normal. No ageusia or anosmia.

CASE XIII. *Traumatic hysteria, right hemiplegia, and hemi-anæsthesia.*—August J., aged thirty years, German, mechanic. Personal history: Patient has had good health, good habits, no nervous disease. Injured slightly by electric wire in August, 1889; received no electric shock but was extremely frightened. Next day headache, vertigo, mental confusion, insomnia, weakness in right arm and leg with tremor. These increased.

Status præsens, October, 1889: Patient is a stout, florid-looking man, has some weakness in right arm and leg; no facial or ocular paralysis.

Sensation—Cutaneous: Patches of incomplete tactile and pain anæsthesia on right forearm and fingers; right leg and foot showed also a slight degree of all forms of anæsthesia. No muscular or joint anæsthesia. Knee-jerks exaggerated, especially on right side; no clonus. Eyes: Vision good; insufficiency of external recti muscles abd. 4, add. 18–20. Visual field limited in right eye, not in left; no amblyopia, color-sense normal. Ears: Slight deafness to watch in right ear, not in left; deafness to high notes in right ear. Smell normal. Taste normal.

A study of the symptoms observed in the foregoing cases shows us that the most constant form of sensory anæsthesia is a limitation of the visual field.

[1] The examination was made for me by Dr. W. H. Bates.

Next in order come disorders of the cutaneous sensations; and of these the pain-sense is oftenest affected, then the temperature- and last the tactile-sense.

The muscular and articular sensibility is rarely involved, and hysterical ataxia is rare.

The color-sense is sometimes implicated, but the complete inversion of the formula was not seen; color-blindness occurred in two cases.

The peripheral field very easily tires for color, so that a patient who at first could recognize a color in the outer limit of the perimeter after a short time ceases to do so.

Limitation of the auditory field for high notes is common, and is usually associated with bone-deafness to some extent. Deafness to low notes is quite rare, but occurred in three cases. The auditory disturbances are much oftener observed in connection with hemianæsthesias.

The pharyngeal reflex is usually absent on the anæsthetic side in hemianæsthetic cases, but it was not absent on the sound side, nor was it absent with the less extensive bilateral anæsthesia of the skin.

The knee-jerk in hysterical hemiplegia was diminished or even temporarily absent. In paraplegia it varied.

Taste and smell were not lost except in the hemianæsthetic cases.

The frequency of the anæsthetic symptoms may be summarized thus:

1. Limitation of visual field.
2. Cutaneous anæsthesia—
   (*a*) Pain.
   (*b*) Temperature.
   (*c*) Tactile.
3. Auditory disturbances: bone-deafness and limitation of field.
4. Dyschromatopsia.
5. Muscular and articular anæsthesia.

I will add a few words now regarding some of the special forms of anæsthesia.

The Cutaneous and Muscular Sensations.—The cutaneous anæsthesias are distributed quite often in zones or patches upon the extremities, and in the glove- or stocking-like distribution. But they are also often unilateral.

The anæsthesia is more marked the more widely it is distributed,

or the more rigidly it is confined to one side. Like hemiplegia, it may be deepest on the arm, and leave the leg and face before leaving the upper extremities. The profoundest types are nearly always associated with some paralysis and tremor, and when the muscular sensibility is involved the paralysis is still more surely present.

These facts all seem to point to a common cortical seat for motor and sensory functions.

The anæsthesia, if unilateral, may be transferred by hypnotic suggestion or cutaneous irritation. I was never able to do this in my cases, however; but I did succeed in removing the anæsthesia over certain areas by the use of metal disks, and this furnishes a test of its functional character.

The greater frequency of the loss of pain-sense in hysteria is, I think, a fact which other experimenters will confirm.

The temperature-sense is more often disordered than actually lost.

The tactile sense is usually a little affected in most cases, but it often remains relatively good long after pain-sense has disappeared.

Hysterical ataxia does, I know, occur; but it seems to be relatively rare and was absent in all my cases. Even in profound types of hemianæsthesia with hemiplegia, the position of the limb may be appreciated by the patient.

In general, it may be said that the hysterical monoplegias and hemiplegias carry with them the profounder sensory disturbances. In hysterical paraplegia often no anæsthetic disorder is discoverable. Such cases are, in fact, rather functional spinal disorders than cortical or "ideal" paralyses like the hemi- and monoplegias.

Anæsthetic hysteria is usually associated also with much mental depression.

A word, finally, in regard to the methods and difficulties of the examinations. In making the tests for anæsthesia in hysterical persons the greatest care has to be taken. Patients are often refractory or soon get tired and become perverse and untrustworthy.

The examination of the skin and deeper sensations does not present so much difficulty. The eye-test, however, requires extra care. It must be remembered that an apparent limitation of the visual field may be due to stupidity or perverseness, or it may be due to refractive errors, to optic atrophy or other organic eye disease. Ziem has asserted that it occurs in diseases of the nose and antrum (*Berl. klin.*

Wochenschr., xxv. p. 37, 1888; *Deutsche med. Wochenschr.*, xv. p. 5, 1889); Moravcsik (*Neurol. Centralbl.*, 1890, p. 230) shows that the visual field may, in hypnotized persons, be enlarged through the influence of cutaneous, auditory, or odorous sensations, and that it may be contracted by depressing, and enlarged by pleasurable emotions.

A point in visual examinations of the field, also of importance, is the intensity of the light. Persons with a contracted field for a white disk have a large field for a bright light; and by using bright colored lights very different perimetric charts will be obtained.

The symptom of contraction of the visual field, therefore, is one whose significance must be closely weighed.

I have made this examination in a large number of neurasthenic persons, and a few epileptics, and in a case of paralysis agitans and Basedow's disease: I did not find any limitation, and the investigations of others also tend to show that it is not found in the neurasthenias or the great neuroses other than hysteria.

There have not been many studies yet of the limitation of the auditory field, particularly for low notes. In testing here it must be remembered that some people are born deaf for high notes, and that in ordinary partial deafness the hearing of high notes may be most impaired.

The test of hearing for low notes has been often made by me, and deafness rarely discovered, but it certainly is present sometimes. Bone deafness usually accompanies the limited auditory field.

In conclusion, I have hoped to bring out some further facts which will give more certainty to our knowledge of the stigmata of hysteria, and enable us to speak with greater confidence of its existence.

The characteristics of hysterical anæsthesia are:

1. Its frequent presence in the retinal field, and its peculiar distribution here.

2. Its distribution on the skin, affecting first the pain nerves, and its modification, disappearance, or transfer by metals, or suggestion, or cutaneous irritants.

3. Its peculiar involvement of the auditory nerve, causing deafness to high and even low notes, as well as its dulling the hearing generally.

4. The rarity of muscular and articular anæsthesia, except in connection with profound paralysis.

5. The involvement of the taste and smell.

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See, also, a very copious bibliography by Thorburn in his work, On the Surgery of the Spinal Cord, p. 225.

DISCUSSION.

DR. S. WEIR MITCHELL: I want to express my pleasure in regard to this study, which ought to be done with many forms of mental disorders with the clearness and closeness which characterize this excellent paper.

The doctor spoke of a paper, I think, in regard to the disturbance of the color-field, and used my name. That is a mistake; it should have been my son's name. It was a paper by him (Dr. John K. Mitchell) and Dr. de Schweinitz, which showed in how many cases there was complete absence of disturbance of the color-field.

It is very common, of course, to see the absence of the sense of pain follow the loss of the sense of touch. There is one case I have under my care of a young person who recovered from a severe hysterical attack, but was left with absolutely perfect sense of touch, but no disturbance of the limit of the color-

field, and with perfect incapacity to feel pain. Stand before her with needles and put them under the nails, in the breast, the nipple, and there would be no sensation of pain. But there was one exception, and I have since found this not uncommon. Although she was able to discriminate between heat and cold with a certain amount of difficulty, if you put anything hot on the skin, and it began to be felt through the skin she had pain. She has been afflicted in this way for several years, and is so to-day.

I want to say when I was in Paris many years ago I saw Burq, and he made three attempts to make transfers for me, and entirely failed in all three cases. I have never seen a complete transfer made.

RECENT OBSERVATIONS IN THE ETIOLOGY AND TREATMENT OF MIGRAINE.

By WHARTON SINKLER, M.D.,

PHYSICIAN TO THE PHILADELPHIA HOSPITAL AND TO THE PHILADELPHIA INFIRMARY FOR NERVOUS DISEASES, ETC.

It has seemed to me well to gather together some of the experience of the past few years in the treatment of migraine, and to cull from the journals and elsewhere what is of value in regard to conditions which may cause it.

This form of headache is of unending interest to the medical profession, for its cure or the alleviation of the paroxysms is a constant problem. Moreover, as Gowers remarks, "The disease is often associated with high intellectual ability and many distinguished men have suffered from it and have supplied more careful observations of the subjective symptoms than we possess of any other malady." We may almost say of it, as Sydenham said of gout, that "More rich men than poor men and more wise men than fools are victims of the affection." Among the prominent men who have had migraine are DuBois-Reymond, Dr. Fothergill, Dr. Lauder Brunton, Dr. Anstie, Sir John Herschell, Sir George Airy, and his son, Dr. Hubert Airy, and we all have among our own medical friends many who suffer from attacks. It is by no means, however, confined to the more prosperous classes, for we meet with it in all conditions of men and at all ages.

The attacks of migraine from uterine disorders have been known to the profession for years, and there is a large amount of literature on the subject. Recently much has been written on headaches from eyestrain, and many of these are of the hemicranic type. Numerous cases have been reported which have been relieved by correcting refraction errors of the eye. The connection between migraine and disorders of vision has been pointed out by several authorities, amongst others, Mr.

John Tweedy, Dr. Savage,[1] Mr. Carter, Dr. William Thomson, Dr. S. Weir Mitchell,[2] and quite lately by Dr. George T. Stevens,[3] whose writings on the relief of various disorders of the nervous system by tenotomy of the ocular muscles have attracted much attention. This author thinks that ocular defects play a conspicuous *rôle* as causative conditions in migraine. He refers to a few cases of "blind headache" in which the fundus of the eye was examined during the period of visual disturbance preceding an attack. The retina has been found pale and brilliant, the optic nerve unusually white, and the main arteries somewhat irregularly contracted in their course. In these cases the field of vision was found to be contracted in a striking manner, in some cases one-half of the field being lost, and in others the central field was gone and imperfect sight remained in the periphery. He finds that, unlike ordinary forms of headache, hemicrania does not easily yield to the simple measure of adapting glasses to correct refraction errors. It is often caused by a complication of refraction trouble and muscular insufficiency. This requires tenotomy and suitable glasses. Mr. Bendelack Hewetson[4] has removed migraine by paralyzing the power of accommodation by atropine. Henry S. Oppenheimer[5] considers eye-strain to be a frequent cause of headache, and believes that it is often overlooked. He relates a case of headache and severe tinnitus aurium cured by the proper application of prisms combined with convex cylinders. He recommends, in the treatment of headaches from eye-strain, first, that all hygienic and medicinal indications be carefully carried out; secondly, a most careful correction of the refraction and accommodation; thirdly, correction of muscular insufficiencies by prisms, beginning with a low degree and, when necessary, increasing in power; and should this prove insufficient, tenotomy.

T. Lauder Brunton[6] speaks of migraine from caries of the teeth. He says:

"The most common causes of headache, indeed, are decayed teeth and irregularities of vision. When the teeth are decayed, rinsing out the mouth

[1] Medical and Surgical Reporter, July 29, 1882.

[2] Ibid., July 25, and Aug. 1, 1874.

[3] Functional Nervous Diseases, by George T. Stevens, M.D. New York, 1887.

[4] The Relation between Sick Headache and Defective Sight. Leeds, 1885.

[5] Headaches and other Nervous Symptoms caused by Functional Anomalies of the Eye. Gaillard's Medical Journal, New York, January, 1889.

[6] Disorders of Digestion. London, 1888.

with a lotion of bicarbonate of sodium, or applying a little cocaine to the exposed pulp, will relieve the headache."

He refers to his own case, in which, during the attack, the pain was limited to a spot in the left temple. On one occasion he accidentally discovered, under the angle of the jaw, a small gland, which was hard and painful to the touch. This led him to examine his teeth, and these he tested by percussing with a steel instrument until he discovered a tender spot on the last lower molar on the left side. He went to a dentist, who found that caries had begun at that spot, but had, as yet, caused no cavity.

He also describes the case of a clergyman who began to suffer from headaches so intense as completely to incapacitate him. He took various medicines in vain, and went for a continental tour, but came back little benefited. Dr. Brunton saw him shortly after his return, and examined his teeth, all of which looked healthy. He then took a steel bodkin and probed and percussed each tooth in succession until he came to one which was tender. The dentist found that this tooth was carious. It was properly filled, and the headaches disappeared.

He further says:

"In cases of headache depending upon a decayed tooth when no toothache is felt, it is not improbable that the irritation in the tooth does not give rise directly to the sensation of pain in the head, but does so by acting through the sympathetic system on the vessels, so as to cause the spasm which leads to the sensation of pain."

J. S. Dixon[1] reports several cases in which the removal of carious teeth stopped attacks of hemicrania.

Dr. Louis Starr[2] speaks of headaches being frequent in children during the period of second dentition. He says:

"Headache is common. The pain is usually temporal or unilateral. It may be seated, however, in the occipital region or in different parts of the face, and sometimes shifts suddenly from the occiput to the temporal region. One or more painful points can often be detected, and generally there is a hard, tender, markedly enlarged lymphatic gland in the submaxillary or cervical region."

[1] Atlanta Medical and Surgical Journal, December, 1856, vol. ii. p. 201.

[2] A Study of the Relationship between the Eruption of the Permanent Teeth and the Ailments of late Childhood. Therapeutic Gazette, April 15, 1890, p. 228.

He suggests that the method of production of the headache is one of direct nervous connection; the submaxillary ganglion acting in the case of the lower teeth and the spheno-palatine in the case of the upper as a medium of transfer of irritation to the vaso-motor nerves.

The nose and ear are now recognized as means by which external irritation may operate in producing headaches. Dr. Brunton[1] finds that in his experience headache depending on disease of the nose is at the top of the head just behind the commencement of the scalp, and headache here should always lead to an examination of the nose.

Dr. Hack, of Freiburg (quoted by Brunton), has observed several cases, both of migraine and of frontal headache, depending upon congestion of the mucous membrane covering the anterior turbinated bones, and has been able to effect a radical cure in several cases by the application of the galvano-cautery to the inflamed and swollen mucous membrane.

Thomas E. McBride[2] relates four cases of migraine which had been cured by relieving the post-nasal catarrh which had existed in each case.

Harrison Allen, in a paper read before the Philadelphia Neurological Society,[3] refers to a patient in whom merely touching the right middle turbinated bone with a probe instantly caused intense vertex pain. He reports a second case in which there were attacks of general headache and clavus over the right parietal eminence. In this patient he found disease confined to the middle turbinated bone on the right side. He also mentions the case of a lady in whom there were frequent attacks of "sick headache" which were dependent upon deep-seated infiltration of the pharynx. The headaches were removed by curing the pharyngeal disease.

John O. Roe[4] has contributed a paper on "The Frequent Dependence of Persistent and So-called Congestive Headaches upon Abnormal conditions of the Nasal Passages." Most of his cases had constant, dull, general headache, but in two or three the attacks were migraine, and were accompanied with nausea and vomiting. In all

[1] Loc. cit., p. 107.

[2] The Medical News, January 30, 1886, p. 136.

[3] On the Headaches which are Associated Clinically with Chronic Nasal Catarrh. The Medical News, March 13, 1886, p. 289.

[4] Medical Record, August 25, 1888, p. 200.

some obstructive disease was found in the nose, and removal of the obstruction was followed by relief from the headaches.

Migraine has been known as a disorder of childhood for many years, and I have elsewhere pointed out its frequency in very young children.[1] I have seen at least one case in which a child of two years had periodical attacks, with every characteristic of migraine. A medical friend has described to me the case of one of his own children who he believes has had attacks of hemicrania from the age of two years. Gowers[2] says that one-third of the cases begin between five and ten years of age.

Russell Sturgis[3] has described a form of recurrent headache in children which he considers to be similar to the migraine of adults, with the difference that the pain is more likely to be across the forehead and is less often confined to either side of the head than that of migraine. He describes these attacks as occurring in children of a nervous temperament, who sleep poorly, with bad dreams and night-terrors, and who are fretful and often chilly and have a heavy expression about the eyes. There were, in many of his cases, optical illusions of sparks or spots, and bands of color. The treatment which he has used successfully in some twenty cases consists in the administration of ten minim doses of the fluid extract of ergot, iron being added if there is coexisting anæmia. The ergot should be continued for several weeks after the cessation of the headaches.

Migraine sometimes begins late in life. A patient, whose case I shall report later, never had an attack until his thirty-eighth year, and Gowers speaks of a man in whom the attacks began at sixty years.

The visual phenomena of an attack of migraine have been discussed for many years. Lately some interesting cases of transient hemianopsia and unusual forms of scotomata have been reported. Dr. de Schweinitz has kindly given me an account of two cases in which the form of scotomata was unusual. In one it was described as being like the wavy appearance of heat rising from a chimney. The cases are as follows:

"A man, aged thirty-two, whose family physician reports general health good, and who, as far as my own examination is concerned, has no organic

[1] Transactions Philadelphia County Medical Society, 1887.
[2] Diseases of the Nervous System, 1888, p. 1172.
[3] Archives of Pediatrics, 1889, vol. vi. p. 293.

disease. For a number of years this patient has had attacks of typical migraine; the invariable prodrome is an appearance as if heat were rising in the air, which is followed by numbness of one hand, or else numbness of the tongue, and, although of this I am not absolutely sure, aphasia. This condition lasts from fifteen to twenty minutes, sometimes as long as an hour, and is succeeded by a terrific explosion of hemicrania. His attacks average one a month, but they have occasionally come as often as once in two weeks. He has a moderately high degree of compound hypermetropic astigmatism, together with insufficiency of the internal recti of eight degrees in accommodation. There are no other muscular anomalies. He is at present under treatment for the eyes.

"The other case is one of a man aged forty-seven, typically neurasthenic, and of bilious temperament, who, for many years, has had severe migraine, the attacks averaging one every two weeks. He has no definite or always-present prodrome, but in a certain number of instances this has consisted of a large, dark, circular object, composed from within outward of a dense black rim, a broader grayish-black circle, a central nucleus of yellow color, and a nucleolus of dense black. It is needless to remark that I, not he, have used these technical terms to describe it, but you observe that this curious appearance which he drew for me contains the components of a compound cell, the dark outer rim corresponding to the capsule, the gray portion to the cell contents, the yellow spot to the nucleus, and the dark spot to the nucleolus. This patient has a high mixed astigmatism, with insufficiency of the internal recti of four degrees."

Dr. Charles A. Oliver kindly referred to me a case of migraine in whom there existed that rather unusual condition of contraction and dilatation of the pupil known as hippus, in connection with the attacks of migraine, and in whom there were other interesting visual phenomena accompanying the attack. The following notes were made by Dr. Oliver:

"*January* 8, 1886. L. L., aged twenty-five years, unmarried.

"For two weeks past she has had a spasmodic action of the right iris, causing the pupil to dilate and contract irregularly, this spasmodic action being felt by the patient as a twitching or a series of movements. During the past eleven months she has had a number of severe attacks of migraine, blindness appearing simultaneously before the right side of each field of vision and quickly moving across to the left side, lasting for fifteen minutes, and followed by clearing up of the visual fields in the same sequence; that is, commencing at the right periphery of each field and ending on the extreme left side. The attacks terminate with the usual signs of headache, nausea, etc. She has had the headaches, which have been worse at the time of menstruation, for several years. She complains of asthenopic symptoms, and she has never

worn glasses. She had four brothers and one sister, she being the third child. A younger brother had a "cast" in his eye, and the mother has worn glasses for years. The patient was overworked, neurasthenic, and her menses were scanty and irregular. Ocular symptoms show:

$$\infty \text{ O. D. V.} = \frac{5}{7}, \frac{1}{2}? \quad \text{Sn. } 1\tfrac{1}{2}\ 6''\text{–}26''.$$

$$\infty \text{ O. S. V.} = \frac{5}{7}, \frac{1}{2}. \quad \text{Sn. } 1\tfrac{1}{2}\ 6''\text{–}26''.$$

"Eyes are slightly *in* under hand-cover.

"The ophthalmoscope showed gray disks with a tortuous condition of somewhat undersized retinal veins, with the characteristic changes of disturbed ametropia. Patient was advised an estimation and correction of refraction error, but she refused to have glasses, thinking that rest from work would relieve the necessity for wearing them.

"*November* 5, 1886. The last attack began as a large, central scotoma, which, she says, was about the size of a silver half-dollar, associated with fronto-temporal pain on both sides and lasting about twenty minutes, followed by numbness of the left upper extremity, commencing in the ring and little finger.

"Patient failed to report for over two years, when she stated that she had been free from attacks until the day when she was seen. This attack appeared as a central, blind area which was not surrounded by any brilliant lines or zigzags. This lasted for about fifteen minutes, and was followed by giddiness, but no numbness. The right-sided hippus was again complained of, and upon careful study was found just as pronounced as at the previous examination. The iris excursions, which were quite extensive, were found to be fifteen times in each half minute upon monocular exposure and but six times in the same length of time upon binocular exposure.

"The patient was next seen April 29, 1890. No attack until three weeks before, when one unexpectedly appeared four days before menstruation. Patient was then highly neurasthenic and nervous."

In June, 1887, Dr. S. Weir Mitchell read at the College of Physicians[1] a paper on some remarkable visual hallucinations, which took the place of the ordinary balls of light and zigzag lines which are so commonly met with as a precursor of an attack of headache. He relates four cases, the first of which is of so much interest that I will repeat it in full:

"Miss W., aged thirty, was in good general health, and able to bear great fatigue, and to use her mind and body incessantly as a teacher. About once

[1] Neuralgic Headache with Apparitions of Unusual Character. Transactions of the College of Physicians of Philadelphia, vol. ix. p. 175.

a week for many years she had attacks of migraine of great severity. When nineteen years old she began, just before the headaches, to see a bright, gold-tinted cloud, and with it an appearance of parti-colored rain. There was most clouding of vision when the sequent pain was over the right eye, and these visual phenomena were not constant. When twenty-eight years old, and still subject to prolonged headaches, the attacks changed their type. After a few weeks of freedom, one day when going upstairs she was abruptly aware of being accompanied on her left by a large, black, and very hairy dog. In some alarm, she ran into a room and sat down, but still found the dog beside her. Being a woman of courage, she put out her hand to touch it, but could feel nothing, although her ghostly companion was still visible. At this moment severe pain began over the left eye, and the dog was gone. The attacks recurred at intervals, as well as the now ordinary brow-pain, without the dog vision, but the 'dog-headaches,' as she called them, were always the most severe. The visual phenomena left her after some years, but, as she went to live abroad, I lost sight of her. I should add, as a curious detail, that nearly always, but not invariably, the dog appeared as she was going upstairs.

"In the second case the apparition consisted in the appearance of a sister who had long been dead.

"The first time it appeared the patient, who was in the fourth month of lactation, was dressing in front of a mirror when she was suddenly aware of the dead sister standing beside her. The vision was followed by a severe pain in the left eye, lasting for many hours, which ended with an attack of nausea and a great flow of pale urine.

"In the third case the form of a near relative appeared just before the attack of headache, and faded as the pain increased, but she was left for some days with a strong desire to kill the person whose image she had seen. The patient came of a highly neurotic family. Two brothers died of epilepsy, and two sisters had been insane. The patient afterward had attacks of melancholia, but ultimately made a complete recovery after removal of both ovaries.

"In the fourth case a man of thirty saw a variety of visions, complex figures of brilliant pink or red, multiple red circles in rapid rotation, which apparently came from the distance. Once there appeared a crescent of silver on the wall, suspended from which were numerous heads in profile. Some were strange to him and some the revival of faces which had long been forgotten. In one attack a red spider appeared which merged into a series of rectangles revolving in swift motion."

De Schweinitz[1] reports six additional cases of migraine with hallucinations of vision which preceded or accompanied the attacks. In one of these patients a large green snake was seen just before the attack. In another, marked visions of animals, always either mice or dogs,

[1] University Medical Magazine, May, 1889, p. 450.

were associated with headache. Once, in the morning, the vision took the form of the water-pitcher in the room, through the handle of which a constant stream of running mice playfully chased each other. Once, in church, the vision was that of a small black dog that curled himself at her feet.

The hallucinations of vision in connection with epileptic attacks have been observed by many authors to resemble markedly those described by Mitchell and de Schweinitz. I have recently seen a patient who had some interesting visual phenomena connected with epilepsy:

Mrs. A., aged forty, has three children, and has had three miscarriages. There is no neurotic family history, and she has never been subject to headaches. From the age of nine or ten years she has attacks in which everything seems far off, or, as she expresses it, "like looking through the large end of an opera-glass." There always appeared in these attacks a face with unpleasant expression, which was very distinct for a time, but gradually faded away. She was not unconscious during the attack, but felt powerless to move or exert herself, and would call to anyone in the room to shake her, so that she might regain self-control. The attacks continued with greater or less frequency until November, 1889, when she had an attack of epilepsy (*grand mal*) in her sleep. Again, in April, she had another attack while driving in her carriage. These are the only attacks of *grand mal* which she knows of having had, and both were preceded by the vision of the unpleasant face.

P. Blocq thinks that ophthalmic migraine should be separated from the other migraines. This variety has pain in the head and visual troubles in the simple form, to which are added at times aphasia and sensory or motor troubles. He reminds us that Charcot has already called attention to the fact that ophthalmic migraine may be prodromic to general paralysis. Therefore, its liability to this serious ending must be borne in mind. Blocq relates three cases of migraine associated with general paralysis, the first of which is a typical one of ophthalmic migraine, reported by Charcot; the second reported by Parinaud, and the third case by himself. In all the cases heretofore described the attacks of migraine had preceded the general paralysis, but in the case reported by Blocq the attacks of migraine accompanied the paresis.

Suckling[1] has reported a case of migraine in a youth of eighteen years, in which the attacks were followed by paralysis of the upper

[1] Brain, vol. x. p. 241.

eyelid on the left side, which lasted forty-eight hours. The patient had been subject to attacks of sick headache since infancy, and the temporary paralysis of the third nerve had occurred since that time. The paroxysms lasted two days, during which time the patient was confined to bed, and was unable to eat. There was a nasty taste in the mouth, and a copious flow of saliva.

The periodicity of the attacks of migraine has been known for many years. It is spoken of by Liveing,[1] but I can find only one writer beside myself[2] who has observed that the attacks may recur on a certain day of the week. Tissot[3] says that Salius relates the case of an Italian monk, who, for three years and seven months, had an attack of violent hemicrania every Monday, the attacks lasting from twenty-eight to thirty hours. Liveing, who refers to Tissot's case, thinks that an exact periodicity like this is probably due to the diet or engagements of the day preceding the attack. This is undoubtedly the case in many instances, but in some of the cases which I have seen there was nothing done on the day previous to the attack different from that done on any other day of the week. I will relate some examples of cases of this kind which I have seen:

CASE I.—Miss C. J. H., aged thirty years, teacher. Menstruation regular, painless. From twelve until twenty-two years of age she had attacks of "regular sick headache." For the past eight years the attacks have been confined to the left side of the head, chiefly in the left brow. The attacks recurred with great regularity, and came always either on Monday or Tuesday. The attacks were often brought on by fatigue or by a draught of cold air. Cannabis indica was given in increasing doses, and the patient was greatly relieved. The periodicity of the attacks was broken up, and the intervals became from eight to ten weeks.

CASE II.—Mr. A. H. W., aged thirty years, temperate, uses tobacco in moderation. He has a sister who frequently suffers from attacks of headache, which come almost always on Sunday.

Mr. W. had sick headaches in childhood, and when about sixteen years of age began to have attacks in which the pain was confined to one side of the head. They occurred almost every Sunday morning for several years. He usually got up with a dull headache, and soon after breakfast had scotomata (red and yellow spots) floating at the upper and external angle of the eye. This was followed by violent pain in one brow or in the occiput. The attack

[1] On Megrim or Sick Headache, p. 36. London, 1873.
[2] Pepper's System of Medicine, vol. v. p. 407. Philadelphia, 1886.
[3] Traité des Nerfs, vol. viii. p. 102. Lausanne, 1788.

would last eight or ten hours, terminating in vomiting or in a sharp attack of diarrhœa, after which he felt entirely relieved. Occasionally the crisis was accompanied by an exceedingly copious flow of urine. The patient usually rose at about 6.30 A.M., but on Sunday lay in bed until 9. This, and the culmination of a hard week's work, probably induced the headache. For the past two or three years his work has not been so hard and his life has been more regular; he now but seldom has a severe headache (not more than three or four in the course of the year), and his general health has become excellent.

CASE III.—Mr. J. D. F., aged forty-one, married, grain merchant. He has had headaches since eight or nine years of age. The attacks were not periodical until five or six years ago, when he became actively engaged in business. He then began to have attacks with regularity every Sunday. They usually came on about midday, without ocular or other prodromes. The pain was always located in the occipital region, and lasted with great severity for five or six hours, when vomiting occurred, after which he was relieved of pain.

I gave him cannabis indica and regulated his diet, and the attacks were very much relieved in frequency and severity. The Sunday attacks recurred for about nine months. He ascribed the attacks to the result of a week's hard work. As he expressed it himself, on Saturday night he would "unbuckle," feeling that his work was done until Monday. Instead of rising at 6, as was his habit, he did not get up until 9 on Sunday morning. He now very rarely has attacks and they are not so severe as formerly.

CASE IV.—Mr. J. M. H., aged fifty, book-keeper, and has close deskwork. Of a nervous temperament. His habits have always been good as to stimulants and he never uses tobacco. He had no headaches in childhood. Twelve years ago, at the age of thirty-eight, after a plain, wholesome dinner, he noticed waves and floating spots before his eyes. These were followed by a severe pain in the head. Since that time he has averaged five attacks in a year. The attacks are very much alike. They are always preceded by scotomata and occasionally there is partial hemianopsia. This is followed by pain over the entire head, which lasts for from twelve to fifteen hours. There is nausea, but no vomiting or crisis of any kind. The attacks almost always occur on Sunday. He rises later on Sunday than on other days, takes a bath about midday, and it is after his bath that he usually has the attack of headache. For some years he has had a permanent scotoma before the eyes. He does not notice it when he is at work, but when he is in a bright light or out of doors the spots are very bright, sometimes irregular in shape, with a double outline, and always seem to be falling.

CASE V.—A. W., aged thirty-three years, single, seamstress. She has suffered from headaches since she was seventeen years of age. For some years her attacks occur about once a week and almost always on Sunday. The attack is usually hemicrania, but the pain is occasionally over both eyes. Sometimes there are intervals of three weeks between the attacks, but

on each of the last three Sundays she has had her usual form of headache. When at the seashore or in more robust health she has had less frequent attacks of headache.

CASE VI.—M. C., aged thirty-five, lady's maid. Both parents are dead. Father died of pneumonia, the mother in confinement. She has one brother and one sister. The brother is healthy. The sister is thirty-eight years of age, and has always had violent headaches. For many years the sister had the attack on Sunday. Her occupation and mode of life on that day or the day preceding seemed to have nothing to do with the attacks.

The patient's headaches began with the catamenial period, and have always preceded the menses. About nine years ago she began to have headaches on almost every Sunday. There was no change in her manner of life at this time, and for three or four years the attacks came with regularity on this day. Then she began to have them on Saturdays, sometimes for two or three months at a time. At present she does not have the attacks so often. She has been free from headache for as long a time as two or three weeks, but she always has an attack before the menstrual period, and does not remember to have ever gone longer than two months without an attack. She has been abroad two or three times, and then she escaped the attacks of migraine for a long period. Sometimes she wakes with pain in the head. At other times the attack begins later in the day. The pain is always confined to one side of the head, and generally the left side. She has no ocular symptoms, except wavy spots like smoke. Occasionally there is vomiting, but not generally. The face and lips are pale during the attack. Her general health is good. Her catamenia are regular and she has no amenorrhœa. Her sleep is excellent, and she is not overworked.

Treatment.—The drugs which have attracted the most attention of late are, undoubtedly, antipyrine, phenacetin, and the host of antipyretic and analgesic coal-tar derivatives, which have been introduced in the past few years. White[1] claims to have first used antipyrine in headache. At all events, it has been very universally employed in every variety of head-pain since its analgesic properties became known. T. S. Robertson[2] has used it in 88 cases of migraine; in 54 the action was satisfactory in the course of from thirty minutes to two hours, and in 15 cases the administration of other drugs was rendered more effective by the use of antipyrine. A negative result was obtained in the remaining 8 cases. He recommends that 22 grains be taken at the onset, and in case the headache continues an additional dose of the same size. Bokenham[3] has used the remedy in 26 cases with

[1] Annual of the Universal Medical Sciences. 1888, vol. iv. p. 444.
[2] Medical Record, May 7, 1887. [3] London Practitioner, February, 1888.

entire success, but, instead of using the large doses usually recommended, he gives only 3 or 4 grains, repeating the dose in an hour, if necessary.

Müller[1] has given phenacetin in migraine and various other forms of headache, but has found that large doses—as much as from 2 to 3 drachms—have been needed to produce good results.

Pesce[2] has used antifebrin with advantage in migraine. P. Guttman[3] uses phenacetin in small doses, and gets as good results as from the use of antipyrine. The great advantage that phenacetin has over antipyrine is that it is much safer, as it does not depress the heart. During the recent epidemic of "grippe" phenacetin proved efficacious in relieving the violent headache associated with that disease.

Rabuske,[4] after trying quinine, arsenic, caffeine, antipyrine, electricity, change of climate, etc., was successful in the treatment of a very bad case of long standing hemicrania by the administration of 8 grains of phenacetin night and morning. The cure was effected after the sixth dose.

Antifebrin has been used quite largely of late. Faust[5] has found this remedy, in doses of ½ to 1 drachm, of great use, the headache being relieved.

A. L. Clark[6] has found that 8 to 10 grains of antifebrin will relieve pain in the head in twenty to thirty minutes. S. Merkel,[7] from an experience of 49 cases of migraine and headaches of like nature, considers this a valuable drug. James Little[8] recommends, in the treatment of migraine, that during the intervals between the attacks the following pill be given twice a day:

℞.—Arseniate of sodium	$\frac{1}{12}$	grain.
Extract of cannabis indica	$\frac{1}{6}$	"
Extract of belladonna	$\frac{1}{3}$	" —M.

He gives, in addition to this, 2 grains of valerianate of zinc twice daily. To cut short a paroxysm he gives 20 grains of the salicylate

[1] Therapeutische Monatschrift, August, 1888.
[2] Le Bulletin Médical, May 30, 1888.
[3] Deutsche med. Zeitung, July 12, 1888.
[4] Ibid., Sept. 13, 1888.
[5] Ibid., June 30, 1887, p. 575.
[6] Chicago Medical Times, Sept. 1888, p. 401.
[7] Munchener med. Wochenschrift, June 12, 1888.
[8] Note on the Relief of Migranous Headache. Transactions of the Royal Academy of Medicine in Ireland, Dublin, 1888, vol. vi. p. 55.

of sodium in a wineglassful of water made effervescent by the addition of a dessertspoonful of the granular citrate of caffeine, a second or third dose to be taken at an interval of two hours.

Nitrate of cytisine (a poisonous alkaloid extracted from the seeds of the cytisus laburnum) has been given by Kräpelin in the angioparetic form with excellent results in two cases.[1] He gives it hypodermically, and was led to use it on account of its power of causing contraction of the bloodvessels. In two cases of the spastic form of migraine in which he used it the symptoms were aggravated.

De Schweinitz and Lewis[2] had a certain amount of success in the treatment of hemicrania with the oil of eucalyptus, and I myself had two or three patients in whom this drug was of marked utility. These authors have lately told me that further investigation has proved that its value is by no means general, although certain cases are relieved by its use. In cases where migraine is associated with the gouty diathesis, treatment of the latter is attended with success as far as relief of the headache is concerned. Haig[3] states that he has relieved many attacks in this form of the disease by giving 20 to 30 drops of dilute nitro-muriatic acid in water, repeated once or twice at intervals of a half hour.

Cannabis indica, which has been given in migraine for many years, still holds a prominent place among the therapeutic agents used in its treatment. For myself, I may say that I consider it of more value in the majority of cases of migrainous headache than in any other headache. It must be given for some length of time and the dose should be increased until slight toxic symptoms are felt. We must remember the great variability in the strength of the drug, and be careful to begin with a minimum dose. I have but recently seen a patient who had marked toxic effects from $\frac{1}{8}$ of a grain of the extract. Seguin[4] several years ago pointed out the benefit of cannabis indica in this form of headache, and insisted on its long-continued use.

Richard Green, who first recommended Indian hemp in migraine,[5]

[1] Neurolog. Centralblatt, Jan. 1, 1888.

[2] The Medical News, July 20, 1889.

[3] British Medical Journal, Jan. 12, 1888.

[4] Paper read before Section on Medicine, New York Academy of Medicine, Nov. 20, 1877.

[5] The Practitioner, Nov. 1872.

has continued to use it with success. He maintains that its effect is not simply palliative, but curative, and that in nearly all cases it gives permanent relief.[1] E. J. Overend[2] believes caffeine to be as complete a specific in migraine as quinine is in malarial fevers. He is himself a victim to the affection. He advises the administration of citrate of caffeine in doses of from 3 to 5 grains as soon as the first indication of an attack is felt, and its hourly repetition until relief is experienced. Electricity is of more or less value, and many cases have been greatly helped by galvanism. I have found this means of marked benefit, but have not depended upon it alone in any case. Labbé[3] has cured a severe case of eight years' standing by thirty-four applications of static electricity. A number of other new remedies have been used to a limited extent in this affection. Among them is exalgine, which I found of use in shortening an attack. Ringer has successfully used tincture of nux vomica in drop doses repeated every half hour.

Amongst the latest remedial agents proposed for the cure of migrainous attacks has been hypnotism. In a work on the subject by Albert Moll[4] he expresses his belief that either post-hypnotic or auto-hypnotic suggestion may be used to cure this disease.

Most authors now agree as to the prime importance of hygienic measures in connection with any remedy used for the relief of this disease. Removal from care and work, with fresh air, good food, and change of climate will do more to relieve the frequency of the attacks than any drug. In connection with this the rest-treatment of Weir Mitchell is of the greatest value, and I have seen many cases of chronic migraine relieved by this means.

DISCUSSION.

Dr. G. Baumgarten: It may be of interest in this connection to relate a therapeutical experiment. In the course of some sphygmographic investigations three or four years ago I was seeing a lady suffering from what she regarded as incurable attacks of migraine. She had a hard, tense pulse, with-

[1] The Practitioner, July, 1888. [2] Pacific Medical Journal, Jan., 1889.
[3] Journal de Médicine de Paris, Nov. 11, 1889.
[4] Der Hypnotismus. Second edition. Berlin, 1890.

out any organic disease to account for it. She was a very spare lady, aged thirty-seven years, of a good deal of intellectual culture, of a nervous disposition, and one of those nervous women who are apt to have that kind of tense pulse. Hers was rather striking, and it was for that reason, I believe, that I tried the experiment of reducing the tension of the pulse. Amyl nitrite had, at one time, given relief during the attack, and lowered the blood-pressure; but it had no permanent effect on either. After some casting about I hit upon pilocarpin, and I succeeded in permanently reducing the tension of the pulse by administering small and frequent doses of it, gr. $\frac{1}{30}$ every four hours, and later, gr. $\frac{1}{20}$ three times a day. For about four months she had no attacks of migraine, to which formerly she had been subject every ten days. After a summer vacation and in the fall she again had attacks of headache. Her pulse was very hard, even after a meal, or a brisk walk. She resumed the pilocarpin, and persisted in the treatment three weeks, and again her pulse became less tense, her attacks of migraine ceased, and I think they have not returned to the present time.

DEMONSTRATION OF PATHOLOGICAL SPECIMENS.

By HAROLD C. ERNST, M.D.,
INSTRUCTOR OF BACTERIOLOGY, HARVARD UNIVERSITY.

I. Bacilli of Leprosy.

Dr. Ernst showed a cover-glass preparation of the discharge from the ulcerations on the arm of a leper lately at quarantine in the city of Boston. The arrangement of the bacilli of leprosy in clumps was well demonstrated.

II. General Tuberculosis of Intestine after Inoculation of Milk from a Suspected Cow.

A rabbit was also shown with general tuberculosis of the intestines, resulting after the subcutaneous inoculation of a few drops of milk from a suspected cow. A child fed on this same milk was dead of tubercular meningitis, and a second—fed on the same milk—was showing the same symptoms. Bacilli of tuberculosis have been found in the lesions, and the post-mortem examination of the rabbit was made six weeks after the inoculation. This rabbit was one out of four inoculated—of which number, three showed the same appearances upon post-mortem examination.

III. Inoculation of "Contagium Vivum" of Vaccine Virus.

Dr. Ernst also communicated the preliminary results of work done by Dr. Stephen C. Martin, of Boston, in an attempt to isolate the actual "contagium vivum" of vaccine virus. These results, as far as tabulated, are as follows: (They are given precisely as Dr. Martin wrote them down. The numbers attached to the calves denote the number in the series of inoculations since the virus was first introduced into this country by the late Henry A. Martin, M.D.)

February	15, 1890.	Inoculated tube No. 1, from vaccine vesicle on calf 1461.
"	20, "	Inoculated calf 1464 from tube No. 1 in two places, producing two vesicles, from which on
"	27, "	Inoculated calf 1465 in three places, producing three good vesicles.
"	26, "	Inoculated tubes No. 2 and W from No. 1.
March	5, "	Inoculated tube No. 3 from No. 2.
"	13, "	Inoculated calf 1467 in two places from No. 3, producing two vesicles, from which on
"	20, "	Inoculated calf 1468 in five places, producing one fine vesicle, two fair, and two showed no reaction. From the first, on
"	27, "	Inoculated tubes x, y, z, and Nelly Lahey, aged two and a half years; Wm. Burns, aged three years. Both had perfect and typical vaccinia. The aureola began at end of eighth day; decided reactionary fever; crust fell on twenty-second to twenty-fifth day. (On May 5th vaccinated Nellie Lahey again with vaccine of proved vigor. Inspected arm on the sixth day, and found effect to be absolutely *nil.*)
"	24, "	Inoculated tubes 1a and 1b from No. 1, and 3a and 3b from No. 3.
"	25, "	Inoculated tube 1a1 from 1a (third generation), and inoculated 3b1 from 3b (fifth generation).
"	27, "	Inoculated calf 1469 from tubes 1, 3, W, 1a, and 1a1. Characteristic vaccinal effects in all.
April	9, "	Inoculated tubes x1, y1, z1 from x, y, z, respectively.
"	24, "	Inoculated calf 1474 in three places from tube No. 3. In two places from tube 3b1 (fifth generation). In two places from tube x1. In one place from tube z. Typical vesicles in one place from No. 3; in both places from No. 3b1. Slight vaccinal effects in the other three places (from x1 and z).
May	1, "	Inoculated calf 1475 from the above. On the eighth day more or less typical vaccine vesicles in all. From which took points and vaccinated children on May 9th.

MAY 11, 1889.

The reasons for not giving a full account of the growth with which the experiments were conducted would seem to be very good ones. The work is not by any means yet completed, and there may be mistakes that later investigations will correct. Too much credit cannot, however, be given to Dr. Martin for the patience and conscientious application that he has brought to the work thus far.

SOME CULTURES OF THE TUBERCLE BACILLUS ILLUSTRATING VARIATIONS IN ITS MODE OF GROWTH AND PATHOGENIC PROPERTIES.

By E. L. TRUDEAU, M.D.,
OF SARANAC LAKE, N. Y.

In connection with Dr. Ernst's most valuable and interesting demonstrations, I would call the attention of this association to some cultures of the tubercle bacillus which I now present, and which illustrate the possibility of bringing about, artificially, important variations in the mode of growth and pathogenic properties of this microbe. Whether the variations hereafter described are due solely to some peculiar conditions of artificial cultivation or to the widely differing types of disease which furnished the original microbes in each instance cannot at present be determined.

Culture No. 1 was obtained at the laboratory of the College of Physicians and Surgeons, in New York, from the lung of a man dead of acute miliary tuberculosis, and is only the third generation direct from the human lung. It is of interest for this reason, and may serve as a standard of comparison to the other cultures. It presents, as you see, on glycerin-agar all the peculiar features which are recognized as characteristic of growths of this microbe, as described by Koch and confirmed by all observers. It grows very slowly in very dry, isolated, and heaped scaly masses, which can be raised entire from the subjacent culture medium without leaving any trace of their presence.

Culture No. 2 was obtained from the lesions of a guinea-pig dead from general tuberculosis induced by inoculation of a small amount of material taken from an old human phthisical cavity. This culture was brought from France by Dr. G. Currier, and has been under cultivation for a long time, being occasionally allowed almost to die out

before being again replanted. Its gross appearance is entirely different from No. 1. It grows much more rapidly in an even film, which, toward the end of the third week, begins to raise in high irregular pleats above the level of the culture medium. It is nowhere dry or scaly, but is moist and of the consistency of very thick cream. It can be scraped clean from the subjacent agar with difficulty.

Under the microscope these two cultures differ only in that the rods in No. 2 are a trifle shorter and broader than in No. 1. They both retain the aniline dyes in the presence of nitric acid with equal tenacity. Inoculated in rabbits, the virulence of No. 1 seems much greater, and the lesions it induces are more widespread and more rapidly fatal to life. Indeed, only one animal out of fifteen was killed within three months by intravenous and intrathoracic injections of culture No. 2, though it produces, at and near the site of injection in the lungs, nodules with a tendency to caseation and containing bacilli, these lesions differing in no appreciable extent from those induced by injection of culture No. 1.

If plants on glycerin-agar are made from the tubercles induced in rabbits by both these cultures, each presents, as it again develops on the culture medium, its own individual characteristics as described above, showing that the variation brought about in No. 2 is apparently not a temporary one, since it can be reproduced again, and each returns to its peculiar type after the microbes have passed through the system of a living animal.

As far as I am aware, this is the first record of any variation capable of being reproduced which has been noted in the tubercle bacillus. In other bacteria, in anthrax notably, wide variations of growth and virulence can be brought about at will; and the interest which attaches to the facts I present is that it is through these variations that a vaccine for anthrax and other microbic diseases has been discovered, enabling the experimenter to produce for those diseases a microbe possessing the protective power of the virulent germ without its deadly pathogenic properties.

EXHIBITION OF CASES.

I. Slow Pulse.
II. Purpura Hemorrhagica Rheumatica.

By D. W. PRENTISS, M.D.,
PROFESSOR OF MATERIA MEDICA AND THERAPEUTICS IN COLUMBIAN UNIVERSITY,
WASHINGTON, D. C.

I. Slow Pulse.

This patient, G. W. P., was presented by me at the last meeting of this Association (see *Transactions*, vol. iv. p. 120). He has continued in much the same condition during the past year. In November, 1889, he had a relapse, of which the following is the record:

Nov. 24, 1889. Pulse 11. Frequent attacks of unconsciousness. During the attacks his pulse ceases to beat for ten, fifteen, and twenty seconds.

25*th.* Pulse 36. Quinine and iron prescribed.

Dec. 1. Pulse 32. Twenty attacks daily.

7*th.* Pulse 12. Pulse irregular for three or four beats, and then a long interval. Fainting attacks are very frequent.

13*th.* Pulse 34. Ammonium bromide and belladonna given.

21*st.* Pulse 12. Fifteen to twenty spells per day.

24*th.* Pulse 36. Spells not so frequent.

31*st.* Pulse 13. Very severe attack of syncope.

Jan. 14, 1890. Pulse 26. Very severe attack of syncope.

15*th.* Pulse 18. For a time thought to be dead.

17*th.* Pulse 18. Better. Temperature 99.6°.

Feb. 6. Pulse 23.

8*th.* Pulse 13. Slight attacks.

9*th.* Pulse 48. At 8.20 A. M. fell on floor unconscious; black in face; no motion for one hour; choking sound in throat. At 9.15 A. M. constant rolling of the head, then jerking, kicking, and bellowing constantly as though in greatest distress. Takes no notice of surroundings, but put out tongue when told to sharply. Nitrite of amyl and hypodermic injection of morphine and atropine.

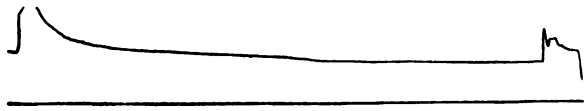
10th. Pulse 32. Recovered from attack of yesterday.
15th. Pulse 24.
21st. Pulse 28. Better and sitting up.
27th. Pulse 28. Swelling of face.
April 8. Pulse 28.

[On May 14th, when the patient was brought before the Association, his pulse was 24. He was apparently comfortable and able to be about, though he still had occasional attacks of syncope.]

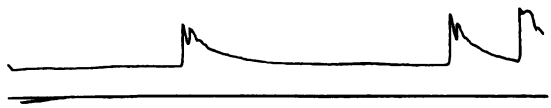
TRACING 1.

Pulse beating 36 per minute.

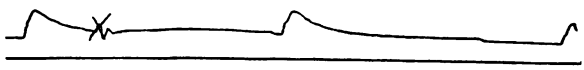
TRACING 2.



TRACING 3.



TRACING 4.



Tracings 2, 3, and 4 taken when pulse was beating 12 per minute. They show the long diastole—5 seconds.

(Tracings were taken by Dr. R. T. Edes.)

II. PURPURA HEMORRHAGICA RHEUMATICA. (PELIOSIS RHEUMATICA; MORBUS MACULOSUS WERHOLFII.)

Eddie D., aged thirteen years. Previous health good. His first attack was in March, 1889, and began with swel ing of all the joints, pain, especially in the knees and ankles, a purpuric eruption over the entire body, and pain in the stomach, and vomiting. The pain in the knees and ankles was excruciat-

ing. He was put on a diet of milk and Mellin's food, and recovered in two weeks. This attack followed prolonged exposure and excitement at the base-ball grounds—the boy, being infatuated with the game, was frequently all day without food, except ginger cakes, pie, and peanuts, and sat on the rough benches of the ball grounds in cold winds, in rain, or in heat.

The second attack was in September, 1889, and was similar in character, except that the swelling and eruption were confined to the right half of the body. He had then been exposed to scarlatina.

The treatment up to this time had been acids, iron, and quinine, and, to relieve pain, morphine.

The third attack was on November 15, 1889, and was similar to the preceding, but more severe. Pain in the abdomen and left side of chest was intense. In addition to the purpuric eruption there were hemorrhages from the bowels and bladder, and, in large patches, into the skin. No hemorrhage from the nose or lungs.

There had been very little fever during the attacks, the temperature seldom rising above 100°. In the third attack he was delirious, and had dyspnœa, swelling of the forehead, and conjunctivitis. Arsenic, which he had been taking for several weeks, was discontinued.

On December 17, 1889, and on February 27, 1890, he had relapses. After this to the present time relapses recurred at intervals of a month or si : weeks, but were less severe. During the interval he is not perfectly well, though comparatively comfortable and free from pain. Since the beginning of the disease the purpuric eruption has never been absent.

The urine was examined from time to time, and, excepting the presence of blood, was found normal. On one occasion, however, there was a greatly diminished amount and much albumin, evidently due to acute nephritis.

Dr. Theobald Smith, Bacteriologist to the Government Agricultural Department, was kind enough to examine the blood, but discovered nothing abnormal, as will be seen by the accompanying report:

"*January* 21, 1890. The blood from the finger was dried on a cover-glass, and stained with alkaline methylene blue. Some of the preparations were decolorized in one per cent. acetic acid. There was no change in the size or form of the red corpuscles. Occasionally isolated corpuscles were observed, which retained the stain faintly. These forms are probably young corpuscles. The leucocytes were slightly increased in number; otherwise the blood elements appeared normal."

Dr. Smith also sent the following interesting note of a hemorrhagic disease that occurs among guinea-pigs:

"Among guinea-pigs, kept for experimental purposes, a disease occasionally appears which usually ends fatally and may carry off the greater number of

those living together. The disease seems to be due to the exclusive use of dry food, such as grain of various kinds. When the food is changed, and vegetables, fruits, etc., are given, the disease is checked and disappears. That it is a food disease I feel quite certain. The examination of the dead animals reveals, as a rule, extensive subcutaneous and intramuscular ecchymoses, limited chiefly to the limbs. Occasionally they are found on or in the muscles of the chest and abdomen. The internal organs contain no bacteria."

The characteristic symptoms in this case of peliosis rheumatica are: Swelling and pain in the large joints, closely resembling rheumatism, but coming and going sometimes within a few hours. The characteristic ecchymoses of true purpura are present. There are hemorrhages into the bladder and bowels, but none from nose or lungs.

Of particular interest in this case was a peculiar hemorrhage into the true skin—apparently between the epidermis and true skin—which destroyed the skin by gangrene. The most marked of these ecchymoses were in the skin of the abdomen—two patches, each the size of the palm of the hand, one on the left, the other on the right of the navel. These ecchymoses appeared within half an hour, and were at the beginning perfectly black and painless patches of true gangrene. In a few days a line of demarcation formed, and in due time the dead tissue sloughed, leaving a granulating surface which healed slowly, forming scars which can still be seen.

The boy formerly had a tight phimosis, for which I intended circumcising him, but one of these sloughs appeared on the under surface of the prepuce and cured the phimosis.

Among the symptoms were frequent vomiting and violent pains in the abdomen. The abdominal pain at times was so violent that I thought there was a peritonitis from a hemorrhage into the peritoneum, but the absence of tenderness and the rapid disappearance of the pain rendered this view improbable.

The attacks, as stated, are remittent in character; exacerbations occurring at intervals of one or two months. The later attacks have been less prolonged than the earlier.

As to treatment, the medicine that appears to have done the most good is arsenic—and possibly salol given for relief of the rheumatic symptoms.

DISCUSSION.

DR. I. E. ATKINSON: It is difficult to say whether these cases of purpura represent any essential disease, or whether they compose a symptom-complex that may be an expression of quite a number of different diseases. Rheumatoid symptoms may occur either in purpura simplex or purpura hemorrhagica, and in either case may fitly be classed as peliosis, or purpura rheumatica. The rheumatic or rheumatoid tendency may extend through the entire series of purpuric disorders, and is marked in that related disorder, erythema nodosum, in which the hemorrhagic element is often pronounced. The abdominal pains noted in Dr. Prentiss's case are quite frequently observed, often associated with evidences of implication of the nervous system to such an extent that one or two authors have proposed, as a further subdivision of the disorder, purpura nervosa. The truth is, that the various forms of purpura shade into each other so gradually that no lines of demarcation can be drawn between them, and the descriptive terms can only serve to designate preponderating characteristics, but not essential differences. Within the past two months I have had under treatment a case of otherwise extremely mild purpura, which could only be considered purpura hemorrhagica in consequence of a most trivial epistaxis, but in which a decided cerebral hemorrhage had occurred with pronounced, but incomplete, hemiplegia. Such results are not extremely rare. A correct conception of purpura admits no distinction, in essential nature, between the most trivial form of purpura simplex and fatal forms of purpura hemorrhagica, or *morbus maculosus Werlhoffii.*

DR. A. JACOBI: I am glad to see that the several forms of hemorrhages of this kind are brought together under one head. I have never been able to find a reason why we should have so many separate expressions in our nomenclature referring to probably the same subjects. We always have to fall back on the disordered condition of the structure of the walls of the bloodvessels. It is not the condition of the blood, but of the bloodvessel walls. As far as the condition of the bloodvessels is concerned, it is probable to my mind we might introduce scurvy under the same head too.

It is only a year ago that a German author contended to have found the bacillus in bloodvessel walls of purpura hemorrhagica. Whether it will be confirmed by further experience remains to be seen. At all events, there must be some change in the structure of the bloodvessel walls. So I have come to the conclusion, if we mean to treat such things, particularly of a chronic character, we have to pay the principal attention to the anatomical structure of the bloodvessels. Then we have to give tissue-builders. We have no better tissue-builders than arsenic and phosphorus. The dose for such a boy as Dr. Prentiss has brought before us would be $\frac{1}{100}$ of a grain of phosphorus three times a day, dissolved in oil or whatever you please, for two or three months or longer. I am positive, in a large number of cases, I have extinguished a ten-

dency to purpura hemorrhagica by such treatment. In the first preparation which has been recommended the phosphorus is probably the efficient factor.

DR. D. W. PRENTISS: In regard to the treatment, I commenced to treat with acids and iron tonics, but it appeared to produce no effect whatever, and seemed to disorder the stomach; and finally, I gave arsenic, which has done more good than anything else. I have not tried phosphorus. I would like to ask Dr. Atkinson, if, in any of these cases, he has found sloughing followed by hemorrhage into the skin? It seems to be a characteristic appearance in this case.

DR. I. E. ATKINSON: I have never observed any case where it has gone through the skin. There has been superficial loss of structure. I believe there are cases on record where such gangrene has taken place.

DR. A. JACOBI: Such sloughs will take place when the hemorrhage is extensive. This boy has scars of sloughing over the larger portion of the abdominal integument. This boy has no heart disease, no enlarged liver; but he has peritonitis. He ought to go to bed and have cold applications made. I think the doctor's diagnosis of peritonitis is a correct one.

DR. JAMES TYSON: I have seen similar sloughing of the skin of the arm in a case under my care.

ACUTE PANCREATITIS.

Report of a Case, and Reference to Recent Contributions.

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Of the several varieties of acute pancreatitis that which is least easy of diagnosis is the suppurative form. This fact is largely due to the tendency of the latter to become subacute or chronic. Even if the early symptoms are sudden and violent, as may sometimes be the case, a quiescent period not infrequently follows, during which no especially characteristic symptoms are to be noted. Although in the course of time an extension of the inflammation to the peritoneum is likely to ensue, the progress of the peritonitis is slow, and shown rather by disturbances of nutrition than by septic symptoms. Furthermore, at the time when the patient comes under observation it may be impossible to obtain an accurate description of the initial symptoms, and an unusually pronounced secondary manifestation may make unduly conspicuous a result and not a cause of the disturbances.

The following case is reported for the purpose of illustrating this difficulty of diagnosis and, at the same time, of calling attention to the minuter changes which are to be seen. The latter will be found to contrast very definitely with those observed in the hemorrhagic and gangrenous varieties of inflammation of the pancreas.

The case occurred at the Massachusetts General Hospital, in the wards of my colleague, Dr. G. G. Tarbell, and I am indebted to him for the opportunity of seeing the patient during life. He has also kindly consented to my use of his notes, from which the following statement of the clinical condition has been compiled.

The patient was a whitewasher, sixty-seven years of age, whose personal and family history threw no light upon the disease for which he was admitted to the hospital, January 2, 1889. His habits were good and there was no history of specific disease.

Five weeks before his entrance he was suddenly seized with a severe pain while at work. He ate a piece of cheese with the hope of relief, but the pain became rather worse than better. He then took two pills and was eased on the following day. Since then there has been no pain. A week later he ate freely of split peas, and, soon after, the abdomen and legs began to swell, a gradual increase in size taking place. About this time he gave up work on account of weakness. He then became jaundiced. There had been neither nausea nor vomiting. There was a lack of appetite for food, but a strong craving for acid drinks. There was neither headache nor disturbance of vision, neither cough nor dyspnœa. Micturition was frequent at night, and the urine was dark colored. He felt well, but desired treatment on account of the jaundice.

When he came under Dr. Tarbell's care he was fairly well nourished. There was intense jaundice of the skin and conjunctivæ, while the lips were rather pale. The tongue had a moist, thick, white coat. The examination of the heart and lungs was negative. The abdomen was distended and tense—tympanitic in the elevated, flat in the dependent portions. There was no fluctuation. The upper level of the hepatic dulness was at the right nipple, and the lower edge of the liver was to be felt in the epigastrium. A freely movable mass was indistinctly felt below the liver, but no connection between the two could be definitely determined. The mass descended on inspiration, and transmitted a pulsation synchronous with the heart-beat. The pulse was 72; respiration 20; temperature 96.2° F. The urine had a specific gravity of 1020, was free from albumin, and contained much bile-pigment.

During the subsequent twenty-nine days of his life the pulse ranged between 60 and 80; the respiration remained in the vicinity of 20, and the temperature, practically normal during the first weeks, became subnormal afterward. He suffered from itching, fulness, and a constant desire for some other article of diet than that he was receiving. He slept much of the time. His weakness steadily increased, and on the day of his death he complained, in thick speech, of a dryness of the tongue and throat. In the afternoon the tongue became pushed upward and backward by a swelling on each side of the floor of the mouth, the breathing became difficult, the pulse feeble, and there was considerable distress. Surgical advice was opposed to any operative measures for relief. The inspiration gradually became more shallow and labored till death took place.

At the post-mortem examination, made twenty-one hours later, the

body was rigid, the skin was of a dark greenish-yellow color. The penis, scrotum, and lower extremities were œdematous. The thoracic organs were relatively free from anatomical changes. The abdomen contained five pints of dark-yellow fluid. The spleen was enlarged, perhaps, one-half, firm, dark red. On section both pulp and blood were increased; structural regions distinct. The prostate was hypertrophied, the bladder trabeculated, the kidneys free from alterations. The liver was slightly diminished in size, of a dark reddish-green color, the surface granular. On section the lobular regions were distinct. The largest bile-ducts were dilated to the volume of the little finger, the smaller tubes correspondingly enlarged, all containing a thin, opaque-gray fluid. About an inch and a half from the duodenal papilla there was a flattened elevation of the wall of the common duct of about a line, over a space a half-inch long. The dilatation of the bile-duct extended from this point inward toward the liver. The common and cystic ducts and the gall-bladder were considerably dilated. The latter held some three ounces of thin, gray fluid. The mucous membrane of the stomach, somewhat thinned, was of a greenish-slate color. There was a diverticulum of the duodenum. The intestine contained mortar-like contents. The pancreas, at first sight, presented no abnormal appearances, and the duct was patent. The obstruction of the common bile-duct was found to be caused by the pressure of a diffused, purulent infiltration of the pancreas at the junction of the head and tail, involving an area as large as a grape. The diseased portion was hardened in Müller's fluid, and I am indebted to Mr. F. B. Mallory, of the Harvard Medical School, for the series of sections in which the microscopical changes have been studied.

The alterations found in the pancreas were circumscribed and of a twofold nature. On the one hand, there was a considerable localized increase and thickening of the interstitial tissue, with extension into the pancreatic lobules, only traces of which were at times to be found. The ducts within this new-formed tissue were dilated. These changes were regarded as having no immediate relation to the fatal illness. On the other hand, there was an extensive, round-celled infiltration, both in the vicinity of the above-mentioned alterations and extending for some distance from them. Within this area were large and small abscesses, frequently so situated, with reference to the ducts, as to suggest their origin from the latter. Ducts were found containing detached epithelium and bacterial colonies. Others were filled with leucocytes, the epithelium being still adherent to the wall. Again, clumps of epithelium were seen in the midst of masses of leucocytes. Ducts were also found filled with pus, while the epithelial lining of the tube was absent, there being no cellular infiltration around the duct, while elsewhere the wall of the duct was formed by a margin of granulation-tissue. At times the abscesses would be found to enter the

lobules, although, in general, the destruction of the latter seemed to be accomplished by the extension into them of the surrounding fibrous tissue. Bacterial clumps were found within and near the abscesses, within a neighboring, hyperplastic lymph-gland, also within narrow, thinned-walled vessels, presumably lymphatics.

In the peripancreatic fat tissue an abundant, round-celled infiltration was occasionally to be seen; an adjoining lymph-gland contained, especially within the follicles, a reddish-yellow, granular pigment.

The pancreatic lobules frequently presented a peculiar appearance which is worthy of note. It consisted in an apparent replacement of the parenchymatous cells by others which contained less protoplasm, and a nucleus stained throughout instead of assuming a ring-like aspect. These clusters of apparently free nuclei were separated by a transparent space from the intervening capillary regions and lay, as a rule, toward the centre of the lobule, and at times occupied the greater portion of its surface.

In contrasting the changes found in this case of suppurative pancreatitis with those seen in the hemorrhagic and gangrenous varieties the following characteristics may be noted: In suppurative pancreatitis —limitation of the lesions, conspicuous affection of the ducts, destruction of the lobules by suppurative and fibrous changes; in hemorrhagic and gangrenous pancreatitis the lesions are diffused, the exudation is hemorrhagic, cellular, and fibrino-cellular, the lobules are destroyed by a coagulation-necrosis.

It will be observed that this case was free from the symptoms which are connected with the severer forms of acute pancreatitis. In the lack of evidence of an epigastric peritonitis of sudden onset, it may be questioned whether the diagnosis of inflammation of the pancreas can be made. The clinical picture here presented is essentially that of an obstructive jaundice, and the death was, presumably, cholæmic. The jaundice was more intense than is usually found in connection with inflammation of the pancreas. The rapidly progressing loss of the patient's strength opposed the theory of a catarrhal jaundice, and the view which seemed best supported during life was that of a tumor obstructing the common duct. The autopsy first revealed that this tumor was an abscess of the pancreas, a cause of jaundice not often kept in mind.

I take this opportunity of calling attention to the contributions to the subject of acute pancreatitis which have been made during the past year, since the delivery of the Middleton Goldsmith lecture on

this subject before the New York Pathological Society. At the same time I am desirous of acknowledging my indebtedness to Dr. E. T. Williams, of Boston, for calling my attention to two cases of earlier date which escaped my research at that time.

The first is reported by Frerichs (*Klinik der Leberkrankh.*, 1861, ii. 382) as one of fatal hemorrhage from rupture of the portal vein.

A man, forty-one years old, given to brandy-drinking, suffered for a long time from disturbed digestion, frequent gastric cramps, persistent diarrhœa, and repeated jaundice. During an attack of cramp of the stomach there was the sensation of tearing in the upper abdomen and the outpouring of a fluid. He soon became very feeble, and fainted. The next day there was complaint of pain in the pyloric and ileo-cæcal regions. The intelligence was clear, the face cyanotic, the extremities cool and pulseless. The abdomen was swollen and tense. Death occurred within sixty hours. The pancreas was large and flabby.

On section, a network of broad and narrow, whitish-yellow streaks was seen, chiefly composed of fat-drops and granules. In the head of the pancreas was a puriform focus of the same nature, near which was a small vein opening into the vena portæ, closed by a red clot. Large masses of clotted blood were in the vicinity. The duct of Wirsung was covered with a soft, fatty layer. There was an extensive hemorrhage, the source of which could not be found, of partly clotted, partly fluid blood, forming a reddish-brown tumor below the liver, and reaching from the gall-bladder to the spleen. The blood was extravasated between the layers of the small omentum, the hepato-duodenal, the gastro-splenic ligaments, and the transverse mesocolon. It extended downward to the kidneys and along the pancreas from the portal vein to the spleen.

Although there must be doubt as to the source of this hemorrhage, it is evident that there was no lesion of any large vessel. So extensive a hemorrhage in this region, in the absence of such lesion, makes it suggestive that the case should be classified among the pancreatic hemorrhages. So long a duration of life as sixty hours after the incipient symptoms is indicative of the existence rather of a hemorrhagic pancreatitis than of a pancreatic hemorrhage, a view which is favored by the symptoms and the suggestion of a fat-necrosis.

A thoroughly typical case is that reported by Habershon (*Pathological and Practical Observations on Diseases of the Abdomen*, third edition, 1878, 338). During life it was regarded as one of cancer of the glands near the pancreas, with secondary implication of the stomach.

The patient was a publican, sixty-four years of age, who, for four years, had suffered from acute attacks of slight pain occurring every three or four months. The pain was in the epigastric and umbilical regions and extended to the spine. Some four months before his death he was suddenly seized with severe pain in the region of the stomach and with vomiting. The latter symptom returned at intervals of three or four days and came on several hours after eating. Five days before his death he was much emaciated, the skin hot and dry, the pulse frequent and sharp. The abdomen was moderately contracted, except at the lower part of the epigastric and umbilical regions, where was a rounded tumor, dull and tender. It was separated by a resonant area from the liver and spleen.

This tumor proved to be the sac of the lesser omentum filled with pus. The pancreas for two or three inches toward the splenic end was healthy, but the rest of the gland formed a black slough about two and a half inches long, detached from the duodenum and lying in a cavity filled with dirty, offensive pus. The gall-bladder contained numerous gall-stones. The immediate cause of death was the result of "the transudation of offensive, purulent serum into the general cavity of the peritoneum."

It is interesting to know that Dr. Habershon regarded this case as one of "disease of the pancreas, suppuration, and gangrene." It presents so many points in common with the fifteen cases of gangrenous pancreatitis previously collected as to leave no doubt of its place in nosology.

Of the contributions to the literature of this subject during the past year, the first is that by König (*Ein Fall von Pankreas-nekrose nach Blutung und Fettnekrose*, Inaug.-Dissert, Kiel, 1889):

A woman, fifty-two years old, eighteen days before her death sought gynecological treatment for a prolapsed uterus. She had suffered for twenty-five years from severe gastric and abdominal pains, at first only in the right, but later extended to the left side. Finally, she also suffered from backache. During the last month of her life these pains became very severe, though at times relieved by treatment. The epigastrium was very sensitive to pressure and the pain did not depend upon the presence of food. It occupied a dull space as large as the palm of the hand beneath the lower, left ribs. There was vomiting and diarrhœa, and the temperature was elevated from one to two degrees. Death took place from progressive loss of strength.

The post-mortem examination was made by Professor Heller, of Kiel. About an inch from the duodenum the pancreatic duct opened, sieve-like, into an irregular, sinuous cavity between the stomach, transverse colon, pancreas, and spleen. From its walls, near the perforated duct, hung necrotic shreds of fibrous tissue containing large quantities of yellow drops, black granules and flakes, reddish-yellow granules and needles—the latter in clus-

ters. Similar dirty-brown, necrotic shreds were attached to the pancreas. The fat-tissue near the stomach, spleen, and kidneys formed a dense, fibrous mass, in which were numerous large and small, rough, white nodules. In the fat-tissue at the lower end of the right kidney these nodules were also of a brown color. The gall-bladder contained numerous calculi. The upper part of the abdominal cavity was walled off by dense adhesions and contained a clear brown fluid of the color of port-wine. In the lower half was an opaque yellow fluid, in which were fibrinous and fibrino-purulent flakes. This acute peritonitis was attributed to ulcers of the vagina, and was regarded as the immediate cause of death.

This case presents an important exception to those previously recorded, since, despite the evidence of a communication between the interior of the pancreas and the surrounding tissues, the characteristic appearances of gangrene are not mentioned. It is also exceptional with regard to the chronic, fibrous thickening of the fat-tissue, in which the nodules of necrosis were found.

A case of hemorrhagic pancreatitis was reported in detail by Dr. W. S. Thayer (*Boston Medical and Surgical Journal*, 1889, cxxxi. 506). Especial interest is to be attached to this communication, since the writer's familiarity with the subject led him to consider throughout that the probable condition was an acute pancreatitis. The urine was tested for sugar, with a negative result.

Langerhans (*Berliner klin. Woch.*, 1889, xxvi. 1114), at a meeting of the Berlin Medical Society, reported a case of pancreatic necrosis which he regarded as due to an acute pancreatitis.

The patient was forty-nine years old, corpulent, not addicted to liquor. He awoke in the morning with headache and dizziness, followed by the vomiting of bile and mucus. The abdomen became markedly distended in the night, and anxiety, dyspnœa, and palpitation followed. On the second day the abdominal swelling diminished somewhat. Constipation, loss of appetite, and severe thirst became apparent. These were very soon accompanied by violent, burning pain in the left hypochondrium and by collapse. On the sixth day he entered Professor Gerhardt's wards in the Charité Hospital with slight fever, swollen, tympanitic abdomen traversed by a faintly depressed, transverse furrow just over the navel. There were small, red, elevated points in the skin of the abdomen, fading on pressure. A diagnosis of typhoid fever was made.

For the next six weeks there was but little change. There was frequent vomiting, but with longer intervals. The vomit had a putrefactive odor in the beginning of the fourth week. At the end of the seventh week the con-

dition became worse. There was mental obtuseness and a feeble memory, suggesting the possibility of a cerebral tumor. A rapid loss of strength then took place, the urine and feces were passed involuntarily, and death occurred sixty-one days after the beginning of the attack, during which there was a loss in weight of forty-two pounds. There was no sugar in the urine.

Behind and below the stomach lay a cavity in which was the necrotic pancreas, completely detached from its surroundings. There were numerous communications between this cavity and the stomach and intestines. The walls of the cavity were trabeculated, the recesses containing a dirty-brown, thick, fluid mass, in which were many grayish-white and yellow fragments. The necrotic pancreas was flabby, soft, easily crushed between the fingers. The head was golden-yellow, infiltrated with biliary coloring-matter. Multiple, disseminated fat-necroses were present in the subperitoneal fat. The intestinal coils were united by fibrinous layers and by older, very vascular bands which contained patches like those in the fat-tissue.

Langerhans stated that, three years before, he had examined another case. It was that of a very strong and fat waitress, addicted to alcohol. She died in convulsions after a short illness. The appearances were similar to those reported above, but there was no communication between the cavity containing the pancreas and the gastro-enteric canal. In the remarks which followed Langerhans's communication, Renvers stated that he had observed two cases in one week, and had made a correct diagnosis in the second case in consequence of the autopsy of the first patient.

Hansemann added that two years ago he had examined, after death, three cases; one was like that reported by Langerhans. In the second the necrosis of the pancreas was less extreme than that of the abdominal fat. In two of the cases a roseola was present, and in one of these the spots persisted after death and overlay necroses in the subcutaneous fat. In a third case the cause was traumatic, the patient being run over by a wagon. Across the upper abdomen the pancreas was sequestrated and lay in a hole full of pus. Numerous necroses of the abdominal fat were present. The patients were all males, and there was no sugar in the urine of any.

Under the title, "A Case of Genuine Acute Inflammation of Pancreas," etc., Dittrich (*Vrtljschr. f. gerichtl. Med.*, 1890, n. F. vii. 43) reports one of hemorrhagic pancreatitis:

The patient, a convict, twenty-one years of age, four weeks before his death suffered from an attack of colic lasting eight hours. He was then apparently

well until within two days of his death, when he was again seized with very violent, abdomnal pain near the transverse colon. During the night he attempted suicide by hanging (possibly on account of the severity of the pain). He was cut down, and on recovery of consciousness suffered from persistent, extremely violent colic until his death in collapse forty-one hours afterward.

The pancreas was friable and extensively infiltrated with blood. The outer two-thirds were transformed into a brownish-black, almost confluent mass. The peripancreatic tissue and that forming the walls of the omental bursa were suffused with blood. The microscopical examination of the pancreas showed a dense, small-celled infiltration of the interstitial tissue, and the parenchymatous cells were granular, with frequent indications of becoming transformed into a granular detritus.

The case of necrosis of the pancreas reported by Dr. H. P. Loomis (*Medical Record*, 1890, xxxvii. 105) presumably belongs to this series

The patient was a man of forty years, weighing two hundred and ten pounds. He had been drinking hard for three months. For two weeks before death he had suffered from diarrhœa, and the stools were at times streaked with blood. A few hours before he died he was mildly delirious, his temperature normal, and the pulse rapid. He then became suddenly and unexpectedly collapsed.

The pancreas contained necrotic areas presenting a gangrenous appearance, which showed under the microscope the characteristics of a coagulation-necrosis. They were considered as most probably resulting from previous hemorrhages into the substance of the organ.

Attention should be called to the report (*Deutsche med. Woch.*, 1890, x. 201) of the meeting of the Hamburg Medical Society, at which specimens of fat-necrosis were shown by Fränkel. They were presented as illustrative of the so-called disease, fat-necrosis, as described by Balser (*Virchow's Arch.*, 1882, xc. 520). The report referred to contains so little of an objective statement of the case under consideration that it can merely be surmised that the disease was an acute pancreatitis. Since there is but little doubt that Balser's cases were of this disease, it is likely that Fränkel's will be found to belong to the same category.

Owing to the intimate relation which pancreatic hemorrhage seems to hold to acute pancreatitis, it is desirable to refer to two cases of the former affection which have been reported during the past year.

The first is by Dr. Harris, one of the medical examiners of Suffolk

County (*Boston Medical and Surgical Journal*, 1889, cxxi. 606). There was superabundant fat-tissue, an alcoholic odor to the breath; death was sudden and unexpected There was hemorrhage into the pancreas and into the adjoining subperitoneal tissue.

Dr. Biggs (*Medical Record*, 1890, xxxvii. 362) also reports a case of sudden death from pancreatic hemorrhage. The man fell dead on coming out of a saloon in which he had taken a few glasses of beer. There was a hemorrhage around the pancreas and a number of minute hemorrhages within the organ.

The literature of the past year thus contains reports, more or less complete, of two cases of pancreatic hemorrhage, two of hemorrhagic pancreatitis, three—probably four—of gangrenous pancreatitis, in addition to the five cases of acute pancreatitis of earlier occurrence and publicly alluded to for the first time by Renvers and Hansemann, a total of ten or eleven cases of acute pancreatitis, in two of which a correct ante-mortem diagnosis was more or less definitely made.

It is evident that this statement is confirmatory of the conclusion previously reached, that acute inflammation of the pancreas "is much more frequent than is generally thought." It is to be hoped that the existence of this disease, which seems securely based upon abundant anatomical and clinical evidence, may soon be strengthened and supported by experimental investigations.

WHAT CAN AND SHOULD BE DONE TO LIMIT THE PREVALENCE OF TUBERCULOSIS IN MAN?

By EDWARD O. SHAKESPEARE, A.M., M.D., Ph.D.,
OF PHILADELPHIA.

Undoubtedly the great majority of advanced thinkers of our profession in Europe and America—such men as those who constitute the body of this Association—to-day admit that Villemin (whatever may have been the reception first accorded his publications) announced a most important truth in 1865 when he declared, from the standpoint of experimentation, that tuberculosis is a specific infectious disease; and unquestionably the same men now acknowledge that Koch, in 1882, not only signally confirmed this truth of Villemin, but greatly multiplied its practical importance by proving that the tubercle bacillus constitutes the sole specific infection of this disease.

It is not my purpose, and, in my opinion, it would waste unjustifiably the valuable time of this meeting, to review even cursorily the evidence which now unshakably supports the claims of these two distinguished investigators. Nor can I hope to present anything new for your consideration.

I think I may safely advance the assertion that there is no fact within the whole range of medical knowledge for the demonstration of which a greater mass of the most exact, positive, convincing proof can be marshalled than that the bacillus tuberculosis is the sole, active, exciting cause of tuberculosis, and that without the agency of this micro-vegetable parasite there can be no genuine tuberculosis, no matter how susceptible the individual or how favorable the so-called predisposing causes may be.

Is tuberculosis at the same time essentially *infectious* and essentially *hereditary?*

Although we have some examples of infectious diseases which are

also at times hereditary, and at least one striking instance of a highly infectious disease—namely, syphilis—which is, perhaps, as frequently transmitted directly to the offspring *in utero* as it is acquired after birth by direct infection, there is strong reason to hold that, as a rule, diseases which are essentially infectious are also essentially non-hereditary.

What relation has this rule to tuberculosis?

The best that can be said of the analysis of clinical experience in favor of heredity is that it utterly fails to account for the origin of 66 to 80 per cent. of the cases of this disease. On the other hand, autopsy records show that tuberculosis *in utero* is the rarest of all affections, in spite of the fact that death from tuberculosis is far more frequent in man after birth than from any other disease. In truth, all the fairly authentic observations of tuberculosis in the human newborn can be counted on the fingers of one hand, and I am cognizant of but two cases in which the lesions have been shown to be genuine and not pseudo-tubercles by the presence of the bacillus tuberculosis or by the test of inoculation. In the bovine species, notwithstanding the great prevalence of tuberculosis among them, such observations are also exceedingly rare.

It seems, therefore, that we have ample ground for the belief that while tuberculosis is essentially infectious it is *essentially non-hereditary*. At least, the "burden of proof" now rests upon those who hold the contrary opinion, and the character of the proof they would offer, let me say, must be unimpeachable and convincing, affording no opportunity for dubious conclusions. Until such proof to the contrary shall be forthcoming there seems to be sufficient warrant at present for regarding the non-hereditary nature of tuberculosis in man as not only highly probable, but as well enough established to constitute a very important factor in determining upon a series of judicious, practical, and comprehensive measures of prevention.

To express thus positively acceptance of the conclusion that tuberculosis in man is essentially infectious and essentially non-hereditary, is by no means to affirm that the influence of heredity has nothing to do in any case with the existence of this disease. That the tubercle bacillus is the sole active infectious agent in the causation of tuberculosis is no more certain than that the influence of predisposition or susceptibility of the invaded organism greatly favors the pathogenic

action of that parasite after it has found a lodgement in the tissues of the body, and that oftentimes it alone makes the development of the implanted seed possible where otherwise a comparatively barren soil or other unfavorable conditions might prevent a fructification. Marked predisposition or susceptibility to the development of tuberculosis upon post-natal infection may undoubtedly be an inheritance transmitted from a tuberculous parent to a child—a transmitted vulnerability which, let us hope, may be within the power of the family physician, through wise counsel respecting hygienic surroundings, wholesome, nutritious food, and judicious physical culture, to do much ultimately to remove.

In recent discussions concerning tuberculosis and the practical value of the lately acquired knowledge of the pathogenic and biological characters of the bacillus tuberculosis we hear much, especially from those chiefly interested in clinical medicine, of the complaint that this great discovery has not yet contributed, and probably never will contribute, anything toward the cure of the disease.

Well, brother clinicians, what if the great Koch and his co-laborers in that new field of etiological research have utterly failed to strengthen your hands by placing therein an efficient means of curing tuberculosis? Cannot a very similar reproach be applied with equal justice to Pasteur and his discoveries concerning the cause of morbid fermentations in beer and wine; concerning the cause of cholera des poules, of charbon, of rouget du porc, and of rabies? Yet, what reasonable man would question the fundamental importance and immense practical value for prophylaxis of Pasteur's discoveries in this direction—because he has not also shown how beer and wine once ruined by the products of morbid fermentation can be restored; or how a chicken seized with cholera, a sheep attacked with anthrax, a hog suffering with rouget, or a human being already in the frightful convulsions of hydrophobia, can be cured? Who would demand of him whom you are convinced comes to you with a new and far more effective means than you now possess of arresting or markedly impeding the spread of an epidemic of scarlet fever, diphtheria, yellow fever, or cholera, that he shall first add something to the treatment of these diseases before his proposed measures of prophylaxis, which you might freely acknowledge to be broadly based upon entirely adequate experimental tests and supported

by the strongest theoretical deductions, shall have at your hands a practical and thorough trial?

In dismissing this point, permit me to suggest that, untenable as such a position would be, it is, nevertheless, very similar ground upon which those stand in this country and elsewhere to-day who, acknowledging the infectious nature of tuberculosis and the tubercle bacillus as the sole active cause of the disease, attempt to excuse or justify inaction upon the valuable knowledge we already possess concerning etiology and prophylaxis by lamenting that Koch and every other bacteriologist have failed to suggest, with their prophylactic measures, an effective mode of therapeutic treatment.

In view of the admitted inefficiency of all present modes of treatment of actual cases of tuberculosis, I hold that effective prophylactic measures are infinitely more important to the general public, and should also be to the physician, as substantial efforts to limit markedly the enormous mortality due to tuberculosis, than can possibly be at the present time the most skilful therapeutic treatment of the disease.

Mr. President, if ever there were reason for quoting the time-worn saying, "An ounce of prevention is worth a pound of cure," there certainly is when discussing the relative importance of treatment and prophylaxis in tuberculosis—a malady which more than decimates the human race and against the ravages of which even the most skilful physician has ever been customarily powerless.

"What can and should be done to limit the prevalence of tuberculosis in man?" This is, in my opinion, one of the most important questions with which we, as physicians, can deal. It is for this reason, and also because it seems to interest the clinician but little at present in comparison to the therapeusis of that disease, that I have selected this subject for presentation before a society formed mainly of clinicians with the hope of provoking a discussion and thereby hastening the time when the clinician shall earnestly unite with the hygienist in an active, incessant warfare for the extermination of that terrible scourge of man—the deadly tubercle bacillus.

Whilst, on the one hand, the discovery of the bacillus tuberculosis has, as far as we can now see, little advanced the clinical management and cure of tuberculosis, except by furnishing a reliable and ready means of early diagnosis, on the other hand it has certainly revealed most important principles upon which to base efficient means of pre-

venting, or at least greatly limiting, the extension of the disease—an end which may be rightly regarded as of far greater moment.

Ernst justly declares that "proper methods of management of tuberculosis, both in human beings and animals, involve more important interests—pecuniary as well as vital—than any other subject that engages the attention of medical men."

What are the general principles which underlie a comprehensive and effective system of prophylaxis of tuberculosis?

I hold them to be essentially these:

1. Destruction of the vitality of the tubercle bacillus wherever it may be found outside the human body.

2. Avoidance on the part of the unaffected of unnecessary risk of introduction within the organism of the living bacillus tuberculosis.

3. Improve the resistant power of the individual, his personal hygiene, and that of his dwelling, in order that conditions unfavorable to the presence, vitality, and development of the tubercle bacillus shall prevail as widely as possible.

I have named these three fundamental principles in what appears to me their order of relative importance, well aware that many clinicians, among them some distinguished members of this Association, look upon the last named as that which alone should receive their hearty sanction and coöperation. I have rated the last as least important for three reasons, each of which I regard as sufficient:

a. Because analysis of the fullest records we have bearing upon the relation of family history to the causation of tuberculosis can, at best, account, through the influence of hereditary predisposition or vulnerability, for little more than one-fourth of the actual cases of tuberculosis.

b. Because, in the first place, the percentage of the so-called predisposed who could be induced to coöperate intelligently with the physician would be small; and, in the second place, it is highly probable that by no means all of even that small percentage could be so built up permanently that every vestige of inherited vulnerability would be removed.

c. Because, even when this inherited susceptibility may be successfully removed by such efforts, we still have to deal with an infectious poison which, if not destroyed, is virulent enough to produce the disease not alone in the comparative few who may be born with an hereditary

predisposition, but even to cause tuberculosis in the great majority who succumb notwithstanding the absence of an hereditary weakness.

I have not thus estimated the value of this means of combating the prevalence of tuberculosis for the purpose of indicating that it should be wholly discarded. On the contrary, I hold that it is incumbent upon us to wage an incessant and relentless warfare against the cause of this disease "with all the means which God and Nature have placed in our hands." But, brother clinicians, let us not indulge the delusive hope that by the selection and even skilful use only of the weakest and shortest ranged weapons we can "successfully cope with so formidable an adversary;" and let us not imagine that when we have done so we have performed our whole duty as physicians.

It is the seed of the disease, without the implantation of which there can be no harvest of death, that we are now most able to reach and destroy; and I hold that both as clinician and hygienist—interested as well in the prevention as in the cure of disease—the true physician should do his utmost to eradicate the seeds of this malady.

It is, therefore, in my estimation, the first two general principles which should form a basis for development of details of the means which can and should be adopted at the present time for the limitation of tuberculosis.

But it has not been my purpose here to suggest in detail the prophylactic measures which should be widely adopted and enforced against the spread of tuberculosis. Excellent regulations—the general observance of which would certainly accomplish a long stride toward the ultimate extermination of this disease, at least in its present pandemic form—have been presented by numerous physicians and health authorities, and the members of this Association are doubtless familiar with them.

My sole object has been to lay this important matter before you in such a manner as to elicit some action, or, at least, expression of opinion, relative to the urgent necessity of doing something, as a representative body of leaders and advanced thinkers in our profession, which shall aim at the limitation of this disease by effective prophylaxis.

Do we admit that tuberculosis is an infectious disease? Then let us so declare. Do we believe that this disease rarely, if ever, has a truly hereditary origin? Then let us not hesitate to publish that belief. Do we acknowledge that the tuberculous, especially in the

active stages of the affection, throw off in the sputa, or in other discharges, the infectious principle of the disease and thereby jeopardize, whether little or much, the safety of the healthy? Then let us boldly publish our knowledge for the benefit of the public, who are not in possession of it. Are we convinced that the meat and milk of tuberculous animals may contain the infectious poison and produce the disease in the healthy consumer? Then let us without further delay strenuously and unitedly demand the rigorous inspection of the meat- and milk-supply and the prompt destruction of the affected animal. Do we agree that habitations become infected with virulent sputum from the consumptive and constitute a danger to the other inmates? Then let us insist upon thorough disinfection and immediate destruction of that dangerous matter. Is it probable that an injured condition of the lungs, or of other important organs, increases the risk of development of tuberculosis after exposure to the infection? Then let us prevent many a case of consumption, by insisting upon the removal of phthisical patients from the general medical wards of hospitals. Are we convinced that the unfortunate subject of tuberculosis constitutes a migrating centre of possible infection? Then let us warmly advocate the establishment of special consumption hospitals.

Now, brother clinicians, I hold that all these things can and should be done to limit the prevalence of tuberculosis in man. But they can be done only through your influence in forming public sentiment by spreading broadcast the knowledge which we possess concerning these vital matters.

DISCUSSION.

DR. E. L. TRUDEAU: I think, perhaps, Dr. Shakespeare has not presented the most encouraging side of Pasteur's work. Pasteur has not only taught us that anthrax and chicken cholera are due to a germ and that hydrophobia is caused by a specific virus which has its seat in the spinal cord, but he has taught us as well that by his methods these diseases are now already somewhat under the control of man, and can be prevented to a great extent. We need not, therefore, necessarily take too gloomy a view so far as tuberculosis is concerned. Dr. Flick has found that the death-rate from phthisis in England has already diminished on account of the establishment of hospitals for consumptives. This should encourage us to build such hospitals, for not only

does experience show that the best results in the treatment of phthisis are obtained in properly located and directed hospitals, but by this means the number of foci of infection is greatly decreased and the death-rate from this disease diminished.

DR. H. A. JOHNSON: I want to say one word as to the habit which we get into of sending our patients away to some locality where consumptive patients are accustomed to go. Within the last two or three years two cases have come under my observation in which tuberculosis has developed in a Western health resort, where up to within the last few years, it has been said, tuberculosis never developed *de novo*. Up to within the last few years it has not been known that tuberculosis has been communicated from the sick to the well in localities where the growth of all vegetable life is at a minimum. I allude especially to Colorado Springs, Denver, and health-resorts in the Rocky Mountains. I have thought it desirable to send patients, not to places where others go with that disease, but, if possible, to places where weeds do not grow well, where plant life does not develop well, for there the bacilli of tuberculosis do not readily retain their vitality. I would like to emphasize the desirability of sending these patients, when possible, to places other than those where tuberculous patients congregate.

DR. TRUDEAU: This plan does not commend itself as either practical or necessary. The tubercle bacillus does not develop outside of the body, and there is no proof that it loses its virulence earlier in the desert than elsewhere. It seems unnecessary that a patient whose saliva, and even nasal secretions, as is frequent with phthisical patients, contain large numbers of bacilli, to say nothing of those which infest his internal lesions, should be sent into desert places in order that he may escape the possibility of infection from the outside air, when this end can be accomplished by the enforcement at health resorts and elsewhere of simple rules with regard to the care of the expectoration. It would seem more practical to teach the laity the rules respecting the danger of dried sputum which are necessary for their protection. I have often seen in the sanitarium under my charge the tubercle bacillus disappear from the expectoration of patients who were improving while they were living daily in contact with fifty other consumptives.

DR. JOHNSON: I do not refer to hospitals for consumptives, but to health-resorts—places where the police of hospitals cannot be maintained. Climatic and other sanitary conditions must, of course, be considered, but these can be found quite as well out of the crowded towns, or, as a rule, perhaps, much better.

DR. TRUDEAU: Aggregation need not be injurious, if proper precautions are taken. I have had charge of a sanitarium for consumptives for the past six years, and not a single employé in it has become consumptive, and many of the patients have been cured. It certainly seems more practical to protect patients in this way than by sending them into the desert. In hospitals, with

care, there need be no sputa on the floor or walks. When I speak of the advantages of putting consumptives in one place, I mean only where proper rules are carried out. I think, in any well-regulated sanitarium these rules can be carried out, and patients can be prevented from spitting on the floor or handkerchiefs. They can be discharged for this. In the Saranac Lake Sanitarium each patient is furnished in each cottage with a paper cup for sputa and these are burned every night, and in this way an enormous mass of expectoration which would accumulate is destroyed. I have seen the tubercle bacillus disappear from the expectoration of patients while they were living in close contact with fifty other consumptives.

DR. JOHNSON: I had no reference to hospitals, but such places as Jacksonville, San Antonio, Denver, and San Diego.

DR. TRUDEAU: We must remember every consumptive carries his own microbes with him. I do not think we expose patients to any greater danger by sending them to a health-resort than they would be exposed to elsewhere in any civilized community, certainly not as much as in the streets of New York.

DR. JOHNSON: The gentleman fails to understand me. I send my patients away, but I desire to send them, whenever possible, away from the centres of population—away from such places as Chicago; and if practising in New York, for the same reasons I would desire to get them away from New York. I know that there is benefit in doing this and in isolation from the large centres where tuberculosis occurs. I have no doubt as to that—no more doubt than I have as to the communicability of tuberculosis. It is not a question of sending them to even such places as Denver, San Diego, Asheville, and retaining them in large cities, but it is as between these places and sending them, when practicable, to some place where they are isolated. For instance, a young man might go on a ranch, go horseback riding, go out on the desert, where there is nothing but sage brush and little of that; he is much more likely to get well in such a case. Even the air seems to be unfavorable to the cultivation of the bacillus. He certainly is less likely to communicate it to others or to receive it. I speak with some degree of experience on the subject, because I have visited most resorts myself. I remember well my first visit to Nice; when I went along the street, there was coughing to my right, coughing behind me, coughing to my left, and a coffin just in front of me, prospectively. I got away from it into the mountains. I was glad to get where the shepherds greeted me, not with their characteristic line from Virgil, but holding out their hands begging for *centissime*. I found there health, and not in the large centres. So my patients have found health. It is not a question between these and hospitals. But you cannot police Nice or San Antonio or any other large place; and I simply desire, in addition to the very sensible, practical, and valuable suggestions contained in the paper, to give this additional caution—when our patients have to be sent away, send them into the open air, as far away as possible into isolated places.

DR. H. C. ERNST: I approve of what Dr. Shakespeare has said, and I think it may be of interest to speak of a case coming under my own observation, illustrating the infection of houses by tuberculous patients, and the transmission of the disease. It occurred in the case of the sister of a servant of mine who went into a perfectly new house near my own. After staying there for perhaps a month she failed so that she was obliged to go home, where she ran down very rapidly, indeed. A sister of hers—my servant—nursed her; the first patient died in two and a half months; the sister, who had been perfectly well until she took up night and day nursing, developed symptoms within ten days after the death of the first, and died in three and a half months. A younger sister, the last of the family, was dead in about six months, under similar conditions; so that in less than a year three sisters were dead of acute tuberculosis. The room which the first patient occupied in the perfectly new house was, after she left, turned into a sewing-room by her mistress—who was not under my care—without any especial prophylactic measures; within eight months this lady was suffering from symptoms of pulmonary tuberculosis. Whilst, of course, there is no scientific evidence that this room was the source of the disease in her case—her family history is good, and her own health was perfect before the occurrence of the first case—there was the focus of infection, and our knowledge of the disease and the way in which it spreads makes it fair to conclude that the source was to be found in that room. Dr. Shakespeare has laid a good deal of stress upon the expressions of clinicians in regard to the work that has been done upon tuberculosis in an experimental direction. I have felt the same lack of sympathy myself in the experimental work that I have carried on; I find not nearly the amount that I think I ought to have. Being a practitioner of medicine, also, I feel very strongly that the methods and practice which one learns in laboratories of the sort of which I have charge are of the greatest possible value in clinical medicine.

The discussion in regard to Colorado Springs has brought to my mind a case which I took there last year. The plans were made for a trip abroad for the young lady and a party. I happened to meet her in the street a short time before they were to sail, and was much impressed by the change I saw in her from her appearance when I had last seen her. As she was going with my sister, and they were to occupy the same rooms, I felt justified in asking for an examination, which I secured with no trouble. There were bacilli of tuberculosis in great numbers in the sputum, and no definite physical signs warranting a change of plans. In less than ten days, however, there was consolidation at the base of the left lung; in two weeks she was in Colorado, and when I left at that time there was every sign of a rapid consolidation of the entire left lung. Since the winter she is reported to have never been in better health. The point of the case is, that it is an illustration of what I believe is an absolute necessity—the routine examination of the sputum for bacilli of tuberculosis, for I feel confident that a majority of cases will recover if the diagnosis and treatment be established sufficiently early.

Another case came to me last winter which also furnishes an illustration of

what may be done by "laboratory methods." The sputum of a patient was sent to me in which I failed to find the bacilli of tuberculosis, but its appearance attracted my attention, so that I wrote asking for an account of the case, what I saw having led me to believe it to be a case of chronic pneumonia. The gentleman who sent the sputum said he was interested to know that I had been unable to find the bacilli of tuberculosis; that the patient had come to him from another physician, who said he had found large numbers of the bacilli, and, on the strength of this, had given a fatal prognosis to the patient, who was perfectly well in less than a month. Evidently, in this case, there had been a mistake in what was seen under the microscope.

At the Massachusetts General Hospital, last summer, I collected a large amount of material, which is not yet examined, but which included a number of cases in which I was tempted to make the diagnosis of pulmonary tuberculosis from the clinical signs. It will be interesting to see how far the microscopic examination will bear out such a diagnosis. I may say that I am unwilling to make a diagnosis of tuberculosis without a thorough microscopic examination. If these points, and others like them, were more thoroughly understood, and if the gentlemen especially interested in clinical medicine would help us a little more, there would be less complaint, as I hear it, that nothing has been done for the cure, or even for the prevention, of tuberculosis.

Dr. Norman Bridge: I am glad to hear all the gentleman has said, and wish to second most heartily the sentiments he has expressed. I am sure that in the last year of practice I have discovered at least half a dozen cases of pulmonary tuberculosis by the microscopical examination of the sputa in which no such discovery could be made by physical exploration. In all these cases, in a few weeks or months, physical signs developed, so that it could be told by physical exploration that some serious disorder more than simple bronchitis was going on in the chest. The time gained by the early microscopical study was the time which, if at all, any particular benefit could be brought to these patients. There is evidence that the lives of some of them have been greatly prolonged, and some, perhaps, saved, by timely resort to proper treatment. I think that it is not only incumbent on clinicians to resort always to the studies that Dr. Ernst refers to, but that the time will come shortly when it will be regarded as malpractice, almost criminal, not to make examinations of that kind.

Dr. E. O. Shakespeare: I am thankful to the gentlemen who have taken part in the discussion. My object in presenting this subject was mainly, as stated in the paper, to call attention of the clinical side of our profession to the vast importance of prophylaxis of consumption as compared with attempts at treatment. Most physicians to-day admit, I take it, that the tubercle bacillus constitutes a most valuable aid in the early diagnosis of tuberculosis—the stage of the disease when therapeutics is at all valuable and offers its greatest power for the limitation of mortality by that means. But while this is a general admission on the part of most intelligent physicians of to-day the world

over, I think, with Dr. Ernst, that however common this belief may be, it is not yet so common that most physicians avail themselves frequently of this mode of early diagnosis. My desire in bringing the subject before the Association was mainly to have it considered from the standpoint of prophylaxis, knowing that however wide the belief in the infectious nature of tuberculosis among the leading thinkers of our profession, yet it is rare to find in any public discussion on this matter, or even in private conversation with the intelligent of that class, a sufficiently strong conviction as to the logical consequences which should follow such a belief. I do not suppose that many members of this Association would now contest the infectious nature of tuberculosis, or that it is the sputa discharged by persons infected with this disease which contain the virulent poison dangerous to those around them; I do not suppose there is one in twenty of our members who would deny that an inflammation of any sort of the bronchial tubes vastly increases the predisposition of any person exposed to the contagion of tuberculosis to contract the disease; and yet what is the almost universal practice in the larger and smaller cities of this country in regard to the management of cases of tuberculosis coming for admission into a general hospital? I know of no general hospital among us where patients known to be suffering even with advanced tuberculosis are excluded from the general medical wards. The infectious nature of the tuberculous material is admitted by clinicians from the standpoint of theory, and, no doubt, there is more or less firm conviction in their minds, based upon their reading on the subject, and in a general way they believe this disease is infectious rather than hereditary. Yet how many are willing, even in their hospital practice, to act on that belief and insist upon the exclusion of tuberculous patients from wards where pneumonia and bronchitis are being treated? How many have the courage in their private practice, when they have under their charge one or more cases of tuberculosis in a family, whether of high or low station, to caution the patients, as well as their associates, against the infectious nature of the discharges which come from the sick, or advise them of the importance of destruction of the virulent principles existing in such discharges before it may be possible for them to be thrown upon the floor or walls and become pulverized and carried up into dust which may be inhaled.

I have chosen this opportunity of enforcing, in the best way I could, the necessity of the practical application of the principles of prophylaxis. To my mind it is the bounden duty of the clinicians not only to give their patients the benefit of the best therapeusis, but, as the guardians of the welfare of the neighborhood in which they live, to publish persistently and advocate strenuously all effective measures of prophylaxis which are known to be based upon satisfactory experimentation and practical tests. I do not know of any city in this country at the present time where the inspection of the meat- or milk-supply is a matter which is even provided for by law. I have seen in the public prints that Pittsburg has made a pioneer effort in this direction, and has determined upon the appointment of inspectors, well paid for the performance of this duty. All honor to her, and may her praiseworthy example

soon be followed by others! Now, there is nothing more certain in experimental pathology than the fact that the tissues of tuberculous animals are infectious; that tuberculous poison, when injected hypodermically into the blood or introduced in the stomach of a healthy animal, is capable, in a large proportion of cases, of producing the disease. There is no better evidence of the fact which has been furnished anywhere in the world than by the papers recently presented concerning that subject by Dr. Ernst, of Boston. Perhaps, no series of experiments relating to this question are more extensive or have been more carefully conducted under the most rigid requirements of pathological research demanded at the present time. They are in accord with other experiments upon the infectiousness of meat, as is well known, and it is not necessary more than to mention the fact. Yet what is the lassitude of the general profession in the cities and elsewhere in this country with regard to the necessity of endeavoring to limit tuberculosis as much as possible by simply insuring the public against tuberculous milk or meat. The questions of prophylaxis which I have briefly presented in this paper I regard, to say the least, as of as much importance as any which the medical profession has to deal with to-day, and they are, I am sorry to have to say, perhaps the most neglected.

Mr. President, what use was it for Koch to have made his discovery of the infectious nature of the bacillus tuberculosis if the practitioners of medicine—those who come in direct contact with the people, who are the natural agents for arousing such a public sentiment as will secure the enactment and enforcement of laws for the protection of public health—utterly neglect to act upon the ample and exact knowledge which we already possess concerning the etiology and prophylaxis of tuberculosis? It seems to me that the treatment of the few cases of disease of this kind which come under the care of the practitioner is of comparatively little importance in the interests of the public, to the seriousness of the duty of the physician to exercise his endeavors to the utmost of his ability to prevent the spread among his neighbors of a disease which destroys one-seventh of the human race, and that despite the most skilful therapeusis possible at the present time.

Dr. Ernst: It seems to me that there should be something said to define what is meant by tuberculosis, and to do away with the statements, as made in books upon clinical medicine, and the implication as well, that all phthisis is true tuberculosis. I think that the formula is one very easily put: that all tuberculosis is produced by this bacillus of Koch, and that all tuberculosis of the lung is phthisis; but the converse does *not* hold true, for *not* all chronic destructive processes in the lung (phthisis) are tuberculosis—it is indisputably proven that some of them may be due to other causes. I have myself been misquoted upon this subject, and I desire to take advantage of the opportunity for placing myself upon record.

MODERN METHODS OF EXAMINATION IN DISEASES OF THE STOMACH,

WITH A CONSIDERATION OF THEIR DIAGNOSTIC VALUE, ESPECIALLY IN GASTRIC CANCER, SIMPLE ULCER OF THE STOMACH, AND GASTRIC HYPERSECRETION.

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As early as 1871 Leube called attention to the advantages to be derived from the use of the stomach-tube for diagnostic purposes in diseases of the stomach. The suggestion introduced the employment of new chemical and physical methods for determining, more exactly than hitherto had been possible, the condition of the gastric secretion, as well as the activity of other functions of the stomach, in its various functional and organic diseases. Since the above date the number of workers in this new field of investigation has gradually increased, until at the present time we are in possession of a very voluminous literature, chiefly German and French, on the subject. Many of the earlier observations, based upon faulty methods and technique, were conflicting and misleading; and yet, through their reappearance from time to time in literature, they continue to obscure the positive knowledge that has been gained on many points in this interesting subject. It is my desire in the present paper to describe, as concisely as possible, those methods which continued experience has shown to be the least liable to error, and to state the conclusions which may fairly be deduced from them.

My personal experience embraces nearly three hundred analyses of the stomach-contents in the past year, besides very many made during a previous period. The basis of the chemical methods employed consists in the withdrawal of the stomach-contents by means of the tube.

The soft-rubber tube is now universally employed, and the form used by Ewald is convenient and practical. It is open at the tip, with one large fenestra, and several smaller ones in its lateral walls immediately above, all the openings having bevelled edges. Its length should be 75 ctm., and for use in dilated stomachs, 95 ctm. Its introduction, as a rule, is very easily effected, and no further lubrication of the tube than that produced by the saliva is necessary. In very sensitive patients it will be found useful previously to spray the posterior pharyngeal wall with a two to four per cent. solution of cocaine. Where the presence of the tube in the stomach excites much retching, the previous ingestion of a small quantity of a similar solution is advised. In cases of suspected ulcer these measures should be employed, at least in the earlier introductions of the tube. Injury to the gastric mucous membrane by the tube itself I believe to be impossible. The gastric contents are withdrawn by the "expression" method, or by aspiration. The former, which consists merely in strong voluntary contraction of the abdominal muscles, sometimes fails in its object, either through lack of intelligence on the part of the patient or through insufficient contraction of weak abdominal muscles.

Before considering the subject of the chemical examination of the gastric contents during digestion it is necessary, particularly in view of a discussion later in this paper on gastric hypersecretion, to refer to investigations on the conditions found in the fasting normal stomach. It has long been believed that the secretory function of the stomach is latent during the fasting period. Rosin,[1] however, as the result of examinations made in 44 fasting patients, believes this view to be incorrect. In only 2 cases could no fluid be withdrawn; in 11 the fluid contained no free HCl; in 31 both free HCl and pepsin were present in the gastric juice, although often its digestive power was apparent only after artificially increasing the HCl percentage. The amount aspirated varied between 3 and 12 c.c. Ewald and Boaz,[2] on the other hand, confirm the experience of Tiedmann and Gmelin, that, under normal conditions, the fasting stomach contains no gastric juice, and suggest that Rosin's results can be explained by the irritation of the gastric mucous membrane by a foreign body, viz., the tube, acting all the more energetically from the stomach being empty.

[1] Rosin: Deutsche med. Wochenschr., 1888, p. 996.

[2] Ewald and Boaz: Klinik der Verdauungskrankheiten, vol. ii. p. 15.

The results of a series of examinations kindly made for me by Dr. Norrie, of New York, in my wards in St. Luke's Hospital, would seem to corroborate the correctness of this view. Ten patients presenting no symptoms of gastric disturbance were examined. The method of procedure consisted in carefully washing out the stomach in the evening and employing aspiration the following morning, the patient fasting in the interval. The time occupied in introducing the tube and aspiration varied from thirty to ninety seconds. In 2 cases no fluid could be aspirated; in 4 cases 1½ to 2 c.c. of a neutral fluid were withdrawn; in 2 cases a few drops of a faintly acid fluid, containing no free HCl, were obtained; in 1 case 2 c.c. containing free HCl, and in 1 case 10 c.c. with free HCl were aspirated. In 2 cases of anæmia, in which 2 c.c. of a neutral fluid were withdrawn at the end of one minute, 15 and 25 c.c., respectively, of a fluid containing 0.11 per cent. and 0.09 per cent. of free HCl, were aspirated by permitting the tube to remain in the stomach three and five minutes.

Test meals of various kinds have been suggested by different authors for the purpose of best judging of the chemical character of the gastric secretion during digestion. Ewald's test[1] breakfast consists of the German semmel, which is nearly equivalent to our white-bread roll, and one-third of a litre of water or weak tea, without sugar or milk. Chemical analysis of the semmel demonstrates the presence of albumen, sugar, starch, fat, and salts. Leube and Riegel prefer a more ample meal, the test dinner consisting of four hundred grammes of water or meal soup, sixty grammes of scraped beef, and fifty grammes of white bread. The advantages of the former consist chiefly in its greater cleanliness and in there being less likelihood of the tube becoming plugged with the partially digested remnants of food. On the other hand, the test dinner undoubtedly excites a more vigorous action of the secretory function. As one or the other variety of meal is selected, it is important to bear in mind certain rules of procedure. The same amount of food, as nearly as possible, should always be given, and the stomach-contents withdrawn one hour after the test breakfast and between the fourth and fifth hours after the test dinner, intervals corresponding to the maximum activity of digestion after the respective meals.

[1] Loc. cit., p. 18.

The basis of all practical conclusions from examination of the gastric contents rests upon a clear recognition of the stages of the normal digestive process which follow the ingestion of one or the other of the test meals described. In repeated examinations Ewald[1] has found the chemical process after the test breakfast to be as follows (my own experience has been a similar one): After the first ten or fifteen minutes the gastric contents have an acid reaction, which is due to free acids, acid salts, or both. The free acid is lactic acid, and this predominates during thirty to forty-five minutes, no reaction being obtained for free HCl by the usual methods. A stage succeeds when an appreciable amount of free HCl is present; the lactic acid gradually disappears and at the expiration of an hour the only free acid present is HCl. These stages are simply prolonged after a more abundant meal, free HCl only being recognizable at the end of the fourth or between the fourth and fifth hours. As Ewald remarks, we are not to understand by this that HCl is not secreted immediately after the entrance of food into the stomach, but rather that it exists in loose chemical combinations, and, moreover, that the presence of salts interferes with the sensitiveness of many of the reagents used for its detection. The latter only permit us to recognize the acid in a free state, and hence it is only at the height of digestion, when saturation of the albuminoids with free HCl has occurred and material for its further chemical combination is exhausted, that we may expect to find more than traces of it.

The non-recognition of the difference between the *secretion* and *presence* of free HCl may be regarded as the source of many conflicting and erroneous conclusions. Any marked variation from the chemical sequence described above may be considered as probably pathological, and upon its demonstration is largely based the clinical value of chemical methods of examination. A certain system in the chemical examination of the stomach-contents is desirable, and I shall, therefore, formulate one which I have found convenient in use.

The gastric contents being first filtered:

A. The reaction of the filtrate is determined.

B. Ten c.c. of the filtrate are employed to estimate quantitatively its absolute acidity.

[1] Loc. cit., p. 21.

C. A small portion of the filtrate is tested for free acids and acid salts.

D. A similar portion is tested for free HCl, and a quantitative analysis of the acid made if necessary.

E. Examination for lactic and other organic acids.

F. Examination for propepton and pepton.

A. By the above analyses we are able to determine the nature of the acidity of the filtrate, the degree of acidity, and finally the existence of peptonizing power. Further examinations consist in testing for rennet ferment, sugar, starch, and its derivatives, which I believe to be seldom of any considerable practical value. A determination, however, of the actual peptic strength of the filtrate is often indicated, as we at present possess no method of estimating the pepton percentage in the gastric contents. I shall consider only those methods for determining the above points which I have found least liable to error.

B. The absolute acidity of the stomach-contents is best determined by titration. Ten c.c. of the filtrate are titrated with a deci-normal solution of sodium hydrate, litmus paper or phenolphthalleïn[1] being used as the indicator. Under normal conditions one hour after a test breakfast, 10 c.c. of the filtrate require from 4 to 6.5 c.c. of the soda solution for neutralization;[2] values above or below this may be regarded as pathological. For the sake of simplicity these values are very generally expressed in percentages of the deci-normal soda solution; for example, by 6 per cent. acidity is meant that 10 c.c. of the filtered stomach-contents require 6 c.c. of a deci-normal solution of sodium hydrate for neutralization. Such an acidity, if due to HCl alone, would represent 0.21 per cent. of this acid.[3] The range of free HCl in the gastric juice in normal digestion is very generally estimated at 0.15 to 0.2 per cent.

C. Leo's test[4] is a simple and convenient one for free acids and acid

[1] Phenolphthalleïn in alcoholic solution has the property of developing a brilliant carmine color in alkaline solutions and of remaining colorless in neutral and acid solutions.

[2] Ewald: l. c., p. 23.

[3] Each c.c. of the deci-normal solution of sodium hydrate contains 7.004 grm. of caustic soda, and will exactly neutralize 0.00364 grm. of absolute HCl. The test alkali is slowly added from a burette, with constant stirring, until a neutral reaction is shown by the indicator. The number of c.c. so used multiplied by 0.00364 expresses the percentage of HCl contained in the 10 c.c.

[4] Leo: Centralblatt f. d. med. Wissenschaft., No. 26, 1889.

salts. A slip of blue litmus paper is moistened with a drop of the filtrate and preserved for a comparison of color. To a few drops of the filtrate powdered calcium carbonate is added and the reaction tested and compared with the original. If the litmus paper is no longer reddened, the acidity of the filtrate is wholly due to free acids; if reddened, though less than before, the acidity is due partly to free acids and partly to acid salts.[1]

D. Examination for free HCl. In Günzburg's phloroglucin-vanillin test we possess one which is at once the most sensitive and the least liable to error of the many proposed from time to time for the detection of free HCl. Many of the color reagents employed for this purpose respond in varying degree to organic acids, and, moreover, their sensitiveness to free HCl is impaired, and under certain conditions destroyed, by the presence of acid salts and albuminates in the gastric contents. These sources of error further explain many of the conflicting statements in regard to the character of the gastric secretion in disturbances of digestion. The phloroglucin-vanillin solution (2 grm. phloroglucin, 1 grm. vanillin, 30 grm. absolute alcohol) in the presence of pure HCl becomes a brilliant cherry-red with the deposition of cherry-red crystals. In the presence of minute quantities of free HCl it is necessary to heat the solution gently, preferably in a white porcelain dish, when, with evaporation of the water, a similar distinct cherry-red color appears in films or streaks at the point of contact of the reagent with the acid solution. In testing the filtrate only a drop or two of the reagent should be added to about 1 c.c. of the filtrate. The above reaction is not developed by organic acids, and, from an extended experience, I believe, does not fail in the presence of albuminates or salts.

The employment of too great heat in testing may result in the development of a brown or yellowish-brown color from the combustion of organic substances, but never in that of the characteristic red color produced by free HCl. The sensitiveness of the test permits of the detection of free HCl in a dilution of 1 to 20,000.

Cahn and v. Mering's[2] method for the quantitative analysis of free HCl has been, and is still, extensively employed for this purpose. The process is a comparatively simple one. It consists in the removal of

[1] The reaction of acid phosphates is not affected by the presence of calcium carbonate.

[2] Cahn and v. Mering: Deutsches Arkiv f. klin. Med., Bd. 39, H. 3, 4; Kinnicutt: New York Medical Record, vol. i., 1888, p. 598.

organic acids from the filtered stomach-contents by distillation and ether and the titration of the watery residue. Honigmann and v. Noorden have shown,[1] however, that the acidity of this residue is not necessarily due to free HCl alone.

Sjoqvist's method demands the facilities of a laboratory.[2]

Leo[3] has very lately published a quantitative test which is apparently more exact and clinically practical. The presence of acid phosphates and lime salts, as well as of organic acids, in the stomach-contents is regarded. The method of procedure is as follows: 1. Remove the organic acids from the gastric filtrate by distillation and ether, and add sufficient water to the residue to restore the original volume. 2. Take 10 c.c. of this residue, add 5 c.c. of a concentrated solution of $CaCl_2$ and titrate with a deci-normal sodium hydrate solution until a faint red color appears, using phenolphthalleïn as the indicator. 3. Take a second convenient quantity, add several grammes of powdered calcium carbonate, and filter. Take 10 c.c. of this filtrate, heat until the free carbonic acid is expelled, add 5 c.c. of concentrated $CaCl_2$ solution, and titrate. The difference in the result of the two titrations will represent the free HCl value in 10 c.c. of the gastric fluid.[4]

E. Examination for organic acids. Uffelmann's test with an aqueous solution of the chloride of iron I believe to be reliable for the detection of lactic acid. The iron solution should be sufficiently weak to be almost colorless. This at once assumes a distinct yellow color in the presence of a *trace* of lactic acid. A few drops of the filtrate are added to the iron solution in a test tube, and, to avoid the possibility of error, I am in the habit of comparing this solution with a control iron solution. Uffelmann's carbolated chloride of iron test for lactic acid, which is so frequently used, is very unreliable, as it reacts

[1] Honigmann and v Noorden: Zeitschr. f. klin. Med, Bd. 13, S. 87.

[2] Sjoqvist: Zeitschr. f. physiolog. Chemie, Bd. 13, Seite 1.

[3] Leo: loc. cit., S. 481–485.

[4] Leo's method for the estimation of the degree of acidity of a solution of HCl in the presence of acid phosphates is based on the fact that calcium carbonate is not decomposed by the latter at ordinary temperatures. Hence, provided the volatile and organic acids be removed, the loss of acidity of the gastric fluid after treatment with calcium carbonate would represent the amount due to HCl, if the presence of $CaCl_2$ in the fluid to be titrated did not determine the formation of a neutral phosphate containing calcium and thus necessitate a double equivalent of the sodium hydrate solution to combine with the chlorine thus liberated. However, the addition of an excess of $CaCl_2$ previous to each titration renders the conditions uniform and the results comparable.

to glucose and certain salts, especially the acid phosphates, which are present, as a rule, in the stomach-contents, as well as to lactic acid.[1]

F. Examination for propepton and pepton. Since propepton is probably a transitional form of HCl pepsin digestion, its recognition in the gastric contents may be at times of practical value. At the maximum of normal gastric digestion only traces of it are found, while pepton is present in abundance; in other words, its presence in large amounts at the height of digestion indicates a deficiency in this function. A reliable test for propepton consists in adding 2 or 3 c.c. of a saturated solution of chloride of sodium to an equal amount of the stomach filtrate, to which are added one or two drops of strong acetic acid. The propepton, if present, is precipitated. The presence of pepton is demonstrated by the so-called biuret reaction. Inasmuch as this is also developed by propepton, the latter must first be precipitated as above described and its filtrate then examined by the biuret test as follows: To 2 or 3 c.c. are added 1 c c. of liquor potassæ and a few drops of a one per cent. solution of sulphate of copper. If pepton is present a *purple-red* color is developed. The chemical examination of the stomach-contents by the methods described, with the exception of the quantitative analysis of HCl, is simple and practical and takes no longer time than an ordinary analysis and microscopic examination of the urine.

The chemical sequence and conditions indicated by the above-described methods in normal digestion, after the test meal, afford a basis for a recognition by the same methods of perversions of the chemical process and the pathological conditions under which they occur. They may be grouped, at least, provisionally, as follows:

I. Free HCl demonstrable at the height of digestion, but apparently in diminished amount; organic acids present; peptonizing power present, but diminished in degree.

II. Free HCl not demonstrable; organic acids present in abundance, usually in excess; peptonizing power sometimes present, sometimes absent.

III. Free HCl apparently increased in amount; organic acids present in small amount or absent; peptonizing power present and active.

[1] By treating the filtrate with ether, which absorbs any lactic acid present, and then testing a watery residue of the ethereal extract with this reagent, the above source of error is eliminated.

The pathological conditions embraced in group I. are chronic gastric catarrh, cachectic conditions produced by various diseases, certain neuroses with apparently depressed gastric innervation, pyrexia of greater or less degree.

II. Gastric cancer, chronic gastric catarrh of a severe type, atrophy of the gastric mucous membrane, amyloid degeneration of the same, toxic gastritis.

III. Simple ulcer of the stomach, secretory neuroses (exalted excitability of secretory nerves), primary and secondary.

The determination of the activity of other functions of the stomach than the purely chemical one is often of diagnostic value

The absorptive power is best estimated by the test proposed by Penzoldt.[1] Two grains of iodide of potassium are administered by the mouth, preferably after a meal, in gelatin capsules (first carefully wiped to guard against any of the salt clinging to their outer surface), and the moment of the appearance of iodine in the saliva noted.[2] Under normal conditions iodine is detected after ten to fifteen minutes. If it appears only at the end of half an hour to an hour the absorptive power of the stomach may be regarded as diminished. It has been found to be defective in gastric catarrh, dilatation of the stomach, etc.

Several methods have been proposed for the estimation of the motor efficiency of the stomach. Of these I shall refer only to the one which I have found the least liable to error, viz., Klemperer's.[3] It consists in introducing 100 grm. of olive oil into the stomach by the tube and withdrawing what remains at the end of two hours. Oil not being absorbed by the stomach, the difference between the amount introduced and that withdrawn indicates the degree of motor efficiency. Under normal conditions the stomach discharges from 70 to 80 grm. into the duodenum in the above interval. I have not observed any clinical impracticability in this method. I have employed it in both hospital and private practice, and believe it to be the most exact of the various methods proposed. Without accepting the extreme view advanced, especially by Jaworski, that the propulsive function of the stomach is its chief one, any impairment of its motor power may be regarded as

[1] Penzoldt and Faber: Berl. klin. Wochenschr., No. 21, 1882.

[2] Filter-paper is saturated with starch paste, dried, and the saliva ejected on it every five minutes. On the addition of strong nitric acid a bluish color is developed in the presence of iodine.

[3] Klemperer: Deutsche med. Wochenschr., No. 47, 1888.

a serious symptom. Klemperer[1] believes that motor insufficiency is not infrequently the *first* symptom of gastric disease, which, later, develops secretory and anatomical anomalies. Its estimation is not only of much diagnostic value, but also of importance in prognosis and therapeusis. In cases of hypersecretion the preservation of motor efficiency implies the absence of ectasia and affords grounds for a more favorable prognosis. In gastric cancer an efficient peristalsis compensates for a time, at least, the perversion of the secretory function. A fair nutrition is maintained by means of intestinal digestion.

It remains to describe the more purely physical methods employed in the examination of the stomach. A recognition of gastric ectasia is often possible only by artificial distention of the stomach with air. Frerichs introduced the method of evolving the gas *in loco* by the successive administration by the mouth of sodium bicarbonate (2–3 grm.) and tartaric acid (1–2 grm.) in solution. Runeberg[2] suggested the introduction of air by means of the ordinary hand-bulb attached to the end of the stomach-tube. Either method is efficient for the purpose. The latter permits of more exactly determining the amount of air necessary in the individual case, which varies very considerably; the possibility of exciting irritation of a sensitive mucous membrane by carbonic acid gas is, moreover, avoided. Distention of the stomach with air has sometimes enabled me to determine more exactly than was otherwise possible the site of a gastric tumor.

Piorry's and Penzoldt's[3] method of determining the lower boundary of the stomach consists in the introduction of a large quantity of fluid by the tube. The lower line of dulness thus produced corresponds to the course of the greater curvature. The occasional absence of gas in the transverse colon may be a source of error in this method. An extension of the lower boundary of the stomach below a line drawn horizontally through the anterior superior spinous processes of the ilium may be regarded as indicating positive dilatation.

Gastro-diaphany, as it is designated by its inventor, Dr. Einhorn, of New York, consists in an illumination of the stomach from within in a manner which permits at least of a recognition of its contour

[1] Klemperer: Verhandlungen des Congresses für innere Medicin., viii. Congress. Wiesbaden, April, 1889, Seite 271–284.

[2] Runeberg: Deutsches Arkiv f. klin. Med., Bd. 34, S. 460.

[3] Penzoldt: Die Magenerweiterung. Erlangen, 1877.

through the abdominal walls. An Edison's incandescent lamp is inserted into the tip of an ordinary soft-rubber stomach-tube, the conductors being carried through the tube and attached to a storage-battery. A dark room is necessary for the examination. The practical use of this ingenious method thus far consists in demonstrating the size of the stomach.

Gastroscopy,[1] direct inspection of the gastric mucous membrane, introduced by Mikulicz, need only be mentioned to be condemned. The introduction of a tube of the size and stiffness necessary for this purpose is painful, and, in my judgment, unsafe.

The prescribed limits of the present paper permit me to consider critically the clinical value of modern methods of examination only in the diagnosis of gastric cancer, simple ulcer of the stomach, and the pathological condition known as gastric hypersecretion.

GASTRIC CANCER.—It is an interesting fact, which Ewald has referred to, that, as early as 1842, Golding Bird,[2] of Guy's Hospital, concluded, as the result of personal investigations, that in the progress of gastric cancer the secretion of HCl was gradually diminished, the percentage of organic acids increasing *pari passu*. The observation was forgotten, but, to-day, as the result of very numerous observations by many of the most distinguished clinicians in various parts of the world, its practical correctness has been corroborated. The secretion of free HCl undoubtedly occurs in many conditions where its presence cannot be demonstrated by the tests at present employed. Such a failure of the tests does not detract from their clinical value, however. Klemperer,[3] while admitting that free HCl is seldom present in the stomach-contents in gastric cancer, doubts whether its secretion is ever diminished. He believes that it is combined with organic bases. Such a combination undoubtedly occurs, and may be accepted as a partial explanation of the absence of free HCl. It is now generally conceded that gastric cancer *per se* is incapable of affecting the secretion of HCl, and by many the failure of tests to detect it in this disease is found in the existence of an associated gastritis and glandular atrophy of varying degree. Pathological lesions of this nature are present presumably only in a slight degree in the case of small and strictly localized neo-

[1] Med. Monatsschrift, November, 1889; Wien. med. Woch., Jahrg. 33, S. 748.
[2] Golding Bird: London Medical Gazette, 1842, vol. ii. p. 391.
[3] Klemperer: Zeitschr. f. klin. Med., 1888, xiv. S. 147–170.

plasms, and under such conditions we should expect to find the secretory function of the stomach but slightly impaired. A case corroborative of this view has been reported by Ewald.[1] In this case free HCl was present in the early examinations, and a sharply localized neoplasm was found on resection of the pylorus, the mucous membrane showing slight changes, except in the immediate vicinity of the tumor. The number of published cases of gastric cancer in which free HCl has been demonstrable is exceedingly small, however. On the other hand, the number of cases in which its constant absence has been observed is correspondingly large. Of these I shall refer only to Riegel's,[2] 16 in number, with 306 examinations, Rosenheim's, and to some personal ones.

In 274 analyses in 13 cases of Riegel's, free HCl was never detected. In his remaining 3 cases, a feeble HCl reaction was obtained only in the early examinations.

In 14 of Rosenheim's 16 cases,[3] no free HCl was demonstrable. In 1, its presence was transitory; in 1, hyperacidity was believed to exist. (The method used for the quantitative analysis in this case has since been found to be fallacious.)

In 8 cases under my personal observation, with 4 autopsies, and in 1 under the care of Dr. Roosevelt, with autopsy, with 132 analyses of the stomach-contents, free HCl was demonstrable only in 2 cases. In 1 of these a trace was detected in 2 examinations out of 12; in the second a feeble HCl reaction was once obtained. In 3 of the cases not confirmed by autopsy, epigastric tumor existed, and repeated hæmatemesis had occurred. In the remaining case no tumor was recognizable, but during a period of two years there had been progressive loss of strength and flesh, and repeated hæmatemesis, vomiting, etc. The patient died of inanition several weeks after admission to the hospital. In 4 of the cases confirmed by autopsy, no tumor was detected in repeated examinations during life. In one of these, a patient seventy-six years of age, there was an absence of all symptoms and signs referable to the stomach, with the exception of complete anorexia and the failure of free HCl reaction to all tests. In the remaining case, in which no free HCl was ever detected in 40 analyses of the stomach-

[1] Ewald: loc. cit., p. 192.

[2] Riegel: Zeitschr. f. klin. Med., 1888, xii. p. 426.

[3] Rosenheim: Berl. klin. Wochenschr., 1888, Nos. 51 and 52.

contents, always after test meals, and in which it was possible to observe the gradual diminution of the peptonizing power until the gastric filtrate was wholly devoid of peptic strength, the neoplasm was localized at the pylorus. Very complete stenosis existed, however, and numerous sections of different portions of the mucous membrane examined by Dr. Thacher, the pathologist of St. Luke's Hospital, showed the presence of a chronic gastritis with glandular atrophy. The anatomical changes varied greatly in degree; in some of the sections no tubules were recognizable, in others they were numerous; small cell infiltration and increase of connective tissue were observed in all sections. There was no regularity in the distribution of the anatomical anomalies. The autopsy was made about six hours after death.

A localized neoplasm, causing pyloric stenosis, may be expected to excite ultimately a chronic gastritis with or without glandular atrophy.

The following conclusions, from investigations up to the present time, seem justifiable:

The diminution or absence of free HCl in chronic gastritis, atrophy of the gastric mucous membrane, and certain other diseases previously mentioned, detracts from the positive value of this sign in the diagnosis of gastric cancer. It is rather to be considered of corroborative value in the presence of other symptoms and signs of the disease.

On the other hand, *the presence of free HCl in the stomach-contents, in repeated examinations, in doubtful cases, is of the greatest diagnostic value, and points very certainly to the absence of cancer.*

Simple Ulcer of the Stomach.—The character of the gastric secretion in simple or round ulcer of the stomach has been the subject of numerous investigations. Riegel[1] has published the results obtained by him in 382 analyses of the gastric contents in 42 cases of this disease. In all the free HCl percentage at the height of digestion was found to be abnormally high. Values of 0.4 to 0.5 per cent. were frequently found. His experience leads him to conclude that hyperacidity is a constant factor in gastric ulcer; that, although it is not necessarily followed by ulcer, it may be regarded as a predisposing cause

Rosenheim,[2] in 8 cases, was able to demonstrate the presence of

[1] Riegel: loc. cit., S. 434.

[2] Rosenheim: Deutsche med. Wochenschrift, No. 22, May 31st.

hyperacidity in only 2; in 4 cases the HCl percentage was within normal limits, in 2 it fell somewhat below. Very lately[1] Rosenheim has published 4 additional cases of ulcer complicated with cancer of the stomach. In all free HCl was present in the stomach-contents; in 2 hyperacidity existed. In the 3 cases in which the diagnosis was confirmed by autopsy the HCl disappeared from five months to several weeks before death.

In 4 consecutive cases presenting characteristic symptoms of this disease (typical gastralgia, repeated hæmatemesis, etc., extending over a long period), personally observed during the past few months, free HCl was found in all the examinations, 31 in number, of the stomach-contents after a test meal. Its percentage in all fell within nearly normal limits, 0.17 to 0.23 per cent. In 1 case, previously observed, in which the autopsy revealed an ulcer the size of a twenty-five-cent piece lying partly within the pylorus and causing extreme stenosis both by hypertrophy of the immediately contiguous structures and by adhesions of the pylorus to the under surface of the liver; hyperacidity was constantly present.

Observations by Cahn, v. Mering, Ritter, and many others, also, would indicate that hyperacidity is not a *constant* factor in simple ulcer. Ewald[2] suggests that Riegel's dictum should be modified as follows: "In gastric ulcer there is secreted a juice constantly containing free HCl and generally in excess."

The constant presence of free HCl, either in normal or increased amount, in simple ulcer of the stomach may justly be regarded as a sign of great clinical value in a differentiation between this lesion and gastric cancer. Especially is this the case in a differential diagnosis between ulcer of the pylorus and cancer occupying the same site. In the former instance the anatomical changes excited in the tissues immediately contiguous to the ulcer, and the not infrequent existence of adhesions, may give rise to an induration recognizable by palpation, and to pyloric stenosis of varying degree. In two cases of ulcer of the pylorus personally observed during the life of the patient, the diagnosis being confirmed by autopsy, a small tumor was easily recognizable and marked stenosis existed.

In cases of cicatricial stenosis from ulcer a tumor, also, may be

[1] Rosenheim: Zeitschr. f. klin. Medicin, January, 1890.

[2] Ewald: loc. cit., p. 232.

present. It existed in three cases personally observed in which the diagnosis, confirmed by autopsy or surgical operation, was chiefly based upon the presence of free HCl in the stomach-contents.

In conclusion, the presence of free HCl in the gastric contents in repeated examinations, in cases of the nature described above, *strongly points to the non-cancerous character of the lesion.*

GASTRIC HYPERACIDITY AND HYPERSECRETION.—The term hyperacidity is commonly used by modern writers to denote an increase in the normal production of free HCl during the act of digestion; hypersecretion to indicate a periodic or continuous secretion of the gastric juice independently of the ingestion of food. To Reichmann belongs the credit of first demonstrating by exact methods the existence of secretory disturbances of this nature. His investigations have been supplemented and corroborated by those of Jaworski, van den Velden, Riegel, Klemperer, and others. Their conclusions, although differing somewhat in details, may be summarized briefly as follows: Aside from the association of hyperacidity with gastric ulcer, hyperacidity and hypersecretion apparently occur both as idiopathic neuroses of the secretory nerves of the stomach and as secondary affections in other neuroses. Hyperacidity frequently exists independently of hypersecretion; hypersecretion, on the other hand, is, as a rule, accompanied by hyperacidity. Hyperacidity and hypersecretion occur in both a periodic and a continuous form; in periodic hypersecretion the acidity often is not increased. Hypersecretion ultimately is very generally followed by atony of the muscular coat of the stomach (motor insufficiency), with all its attendant evils.

Jaworski[1] believes these secretory disturbances to be of very common occurrence. He found hypersecretion in 121 out of 222 gastric cases examined, and in 115 of the 121 there was associated hyperacidity. Jaworski includes all cases under the head of hypersecretion in which more than 10 c.c. of gastric juice can be aspirated from the fasting stomach. Riegel's[2] investigations also indicate the frequent existence of hypersecretion. Ewald,[3] on the other hand, in 600 patients observed this condition only 15 times. He states, however, that he is incompetent to speak authoritatively on the subject, as he

[1] Jaworski: Wiener med. Wochenschr., Nos. 49–52, 1886.
[2] Riegel: Zeitschr. f. klin. Med., 1887, Bd. xii. S. 426.
[3] Ewald: loc. cit., p 423.

has examined for hypersecretion exclusively in those cases presenting its most characteristic symptoms. My own experience, with similar limitations, is closely in accord with Ewald's.

Inasmuch as the question of the condition of the fasting stomach is still *sub judice*, in order to avoid any possible error I have fixed as high a limit as 50 c.c. of gastric juice as a basis for a diagnosis of hypersecretion. I have been able to demonstrate it in three patients whose symptoms suggested its existence. Its early recognition is very desirable, as by appropriate treatment the sufferings of the patient can be greatly relieved and the affection itself possibly controlled. The history and symptoms of a patient are of assistance in a diagnosis of hypersecretion, but positive proof of its presence is furnished by modern methods of examination.

Hyperacidity and hypersecretion have been observed in cases of melancholia, hysteria, neurasthenia, and in other neuroses; hyperacidity as a temporary symptom in the gastric crises of tabes dorsalis and as a transient symptom accompanying or following the passage of gall-stones and renal calculi. The symptoms are of a nature to be expected from an irritation of the gastric mucous membrane by a hyperacid secretion. They are epigastric sensitiveness and pain, pyrosis, and eructations; if the hypersecretion has been of long standing the symptoms and signs of gastric ectasia are superadded. The tongue is usually clean, the appetite is rarely diminished and is frequently increased; there is often excessive thirst. The pain, which usually begins at the height of digestion and continues during the fasting period, is frequently very acute and paroxysmal, the patient often crying out in the attacks and assuming a sitting posture with the body bent forward. Relief is often obtained by the ingestion of warm fluids, particularly milk, but not infrequently only by the use of opium. The digestion of carbo-hydrates is delayed, while that of the albuminoids is rapidly effected. In the early stages the general nutrition is usually unimpaired, though anæmia is often present.

Suggestive as the above symptoms are of hypersecretion, a positive diagnosis is only possible by the following method of examination: The stomach should be washed out, preferably in the evening, and the tube again introduced the following morning, the patient fasting in the interval, and expression, or aspiration employed. If hypersecretion is present, a clear or more or less turbid fluid, colorless, or more

commonly of a greenish or greenish-blue color, sour smelling, of a strongly acid reaction, and varying in amount from 50 to 1000 c.c., is withdrawn. This fluid, in the absence of any fragments of food, is devoid of organic acids and pepton, usually contains free HCl in larger amount than normal gastric juice, and digests coagulated albumen rapidly; in brief, it is hyperacid gastric juice. The following case furnishes a typical example of this disease:

E. H., aged fifty-eight, dressmaker, admitted to St. Luke's Hospital October 7, 1889.

Previous history: Good health until four years ago. Digestive disturbances since. For the past eighteen months epigastric pain and distress, appearing about four hours after meals, relieved at first by voluntary vomiting. The vomited matter rarely contained food, was copious, acid, and had the appearance of dirty water. For the past eight months the attacks of pain have been very acute, and unrelieved by vomiting, and have occurred as frequently during the night as during the day. Partial relief was obtained by the frequent ingestion of large quantities of warm water or milk, sometimes by lying on the stomach, frequently only by the use of some medicine the nature of which is unknown. The patient was incapacitated for work.

Condition on admission: The above-described symptoms were present. Physical examination of the heart, lungs, and liver was negative. The spleen was slightly enlarged. The examination of the urine was negative. Examination of the blood: red blood-corpuscles, 4,131,250 per c.mm.; hæmoglobin percentage, 44. The stomach was dilated; the lower boundary, both by inflation and by the water test, was recognizable on a level with a horizontal line one and a half inches below the umbilicus. Its absorptive power, determined by the iodine test, was nearly normal. Its motor power was diminished; two hours after the introduction of 100 c.c. of olive oil 60 c.c. were recovered. One hundred and thirty c.c. of a slightly turbid, greenish, highly acid fluid were withdrawn by aspiration from the stomach in the morning, careful lavage having been employed the previous evening, the patient fasting in the interval. The fluid contained no organic acids, acid salts, or pepton; it digested coagulated albumen rapidly; free HCl (0.23 per cent.) was present. During a period ending January 1st, thirteen examinations of the contents of the fasting stomach were made with very similar results. The amount of gastric juice withdrawn varied from 75 to 130 c.c. on different occasions; the percentage of free HCl was always above normal.

Daily lavage and large doses of sodium bicarbonate, given systematically after meals, and repeated in two hours, gradually procured entire immunity from the attacks of pain, and the patient gained slowly in weight and strength. The degree of hypersecretion varied but slightly, however. In the latter part of January the patient suffered from a very severe attack of zoster. From this date until her discharge from the hospital, on March 25th, 26 examina-

tions were made: 13 of the contents of the fasting stomach, 13 of its contents after a test meal. In 7 of the former the fluid gave a neutral or alkaline reaction; in 6 it was slightly acid, containing 0.06 to 0.08 per cent. of free HCl. The amount of fluid obtainable by aspiration varied from 48 c.c. to (on one occasion) 115 c.c. The patient continued to be free from all epigastric pain and distress, and was discharged, having gained nine pounds in weight. All medication was dispensed with during the last month of residence in hospital. Daily lavage was, however, employed. The percentage of free HCl in the gastric contents after a test meal, from March 1st, was never above normal. The final disappearance of hyperacidity nearly coincidently with the development of the zoster, the medication in no respect being changed, is an interesting fact.

In a second case of hypersecretion with gastric ectasia, under my observation, the sufferings of the patient, which were very acute, were temporarily relieved by a similar method of treatment. The patient left the hospital too soon to judge of its further results. A third case is still under observation. The patients whose cases have been referred to were all of a distinctly neurotic temperament.

Modern methods of investigation in diseases of the stomach are fairly entitled to the credit of a demonstration of a pathological condition which previously had only been suspected. The purpose of the present paper has been fulfilled if the most exact and practical of these methods have been clearly described and their true value for diagnostic purposes indicated. Their value in suggesting a more exact therapeusis has only been incidentally referred to.

In conclusion, I desire to express my indebtedness to the gentlemen of the house staff of St. Luke's Hospital, Drs. Van Horne Norrie, Colman Cutler, and Levi Warner, and to Dr. Robert Devlin, of New York, for their efficient assistance in my investigations.

METHODS OF DIAGNOSIS IN DISEASES OF THE STOMACH.

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A NEW era may be said to have been inaugurated in the study of diseases of the stomach by the introduction of the Nélaton catheter, which, adapted in size and length to the stomach, renders it now possible to subject the organ and its contents to methods of examination formerly so uncomfortable, and often even so dangerous as to prevent their general employment. Before the sound came into common use, the diagnosis of gastric diseases rested almost exclusively on rational signs. The sound gives us now means of physical diagnosis. What results have been won by the large amount of work done in this direction the past eight or ten years?

There are two main divisions into which the physical diagnosis of diseases of the stomach naturally falls:

1. Secretion and its modifications.

2. The length of time required for digestion; propulsion and absorption; and the capacity of the organ.

1. *Secretion and its modifications.*—It is obvious that the chances of error will be diminished if uniformity is observed in the method of preparation of the stomach for the application of tests. All are agreed that uniformity is desirable, but all are not agreed as to the best method to pursue. The test breakfast is favored by some; the test dinner by others. Ewald holds that a breakfast of tea and dry bread is sufficient to call out the full activity of the stomach within one hour. Others maintain that such a meal involves two opposite sources of error: its lightness may make digestion appear better than it really is, or may, on the other hand, demand so little as to make digestion appear more feeble than it really is. By these critics a heartier meal, com-

prising albuminoids, and a later withdrawal are recommended. Here at the very outset we encounter divergence of opinion as to a point which is probably of minor, perhaps of no importance, but of precisely how much we are not as yet sure. Ewald's method is the more convenient, on account of the less liability of the tube to become blocked by large bits of food.

Perhaps the most important constituent of the gastric juice is hydrochloric acid; certainly none other has received so much attention. It is now generally held that at the height of ideal digestion this acid ought to be present in the free state in about the proportion of two-tenths of one per cent., and that there should be no other free acid. Within a few years we have seen a succession of clinical tests for this acid in its free state proposed and abandoned. About a dozen are given by Jaksch in the second edition of his *Klinische Diagnostik*. Methyl-violet, tropæolin, Congo red, had their day one after another; but it was found that no one of them was satisfactory, the desired reaction being often masked or simulated by other substances. The phloroglucin-vanillin test seems to be decidedly more trustworthy. With the benzo-purpurin test I have no personal experience. Jaksch classes it with the phloroglucin-vanillin, but considers that none of them is always reliable. If a positive reaction is obtained with the two latter, the presence of free hydrochloric acid may be confidently inferred; but albumin, peptones, and salts in considerable amount, may, it is said, prevent its appearance though free hydrochloric acid is present. With the estimation of the amount of this free acid the clinical difficulties are still greater. The titration method, with caustic soda and phenol-phthaleïn or litmus, is practical enough provided the organic acids are absent. Lactic acid is sought for by Uffelman's test, but there are here notable sources of error; and the accurate method of extraction by ether is so troublesome as not to have general clinical value. The fatty acids offer no great difficulties. Pepsin and the rennet ferment seem to go so closely hand-in-hand with free HCl as greatly to simplify the clinical work, provided that the tests for free HCl are worthy of confidence. If, therefore, HCl is absent or diminished, so are apparently pepsin and the rennet ferment; but pepsin at least may be diminished though the inorganic acid is increased.

If we grant, as I do not feel sure that the experience of the next ten years or so will warrant us in doing, that we now have methods at

our disposal which enable us to determine clinically with comparative ease and considerable certainty the presence or absence, and, though with less ease, the amount of the chief chemical agents of gastric diges tion, if we grant that, in the application of the results of these tests to diagnosis our difficulties increase, as it seems to me. Let us consider for a moment the changes which opinion has undergone the past few years as to the significance of the absence of free hydrochloric acid. How brief is the time since it was heralded abroad that a pathognomonic sign of gastric cancer had been discovered in the absence of free HCl! Then it became necessary to admit that the free acid and cancer may coexist. Gradually we have come to know that the absence of free HCl is indicative rather of the extent than of the nature of processes involving the mucous membrane; and the fact also appears, to me very significant, that the acid and its natural concomitants may be persistently absent for weeks and months in nervous dyspepsia, *i. e.*, simply as the result of faulty innervation—at least so Ewald assures us. We see, therefore, that the presence or absence of this acid, even when persistent, is not in and by itself diagnostic of anything at present.

There is another source of error which, as it seems to me, may not have received sufficient attention. I refer to the possibility that in certain individuals, or in any person at certain times, there may exist a secretory gastric inadequacy, so to speak. HCl, pepsin, and the rennet ferment may be formed, though not in sufficient amounts to leave a free excess. Again, it is conceivable that at times, or in certain persons, these digestive agents, secreted in normal amounts, should enter more rapidly than is quite normal in combination, and consequently not be found in the free state. Soluble chlorides and peptones are probably rapidly absorbed from the gastric mucous membrane, and we have as yet no measure of the amount of HCl and pepsin formed; we have only approximative clinical measures of that which remains free.

With hypersecretion and hyperacidity my personal experience is less than with the other branches of gastric chemistry, and I therefore avoid any discussion of these points.

2. As to *the duration of digestion*, it seems to be pretty generally conceded that a healthy stomach should be free from food seven hours after a hearty meal. Whether this limit is correct or not, the length

of time required by any person to empty his stomach of food can be determined accurately by means of the sound, and this determination may be of considerable importance. An undue rapidity of digestion can be due probably only to one of two things—hyperacidity or increased peristalsis, unless we also take into account a vertical position of the stomach. The latter is rare, while of primary pathologically increased peristalsis we have no absolute knowledge. If digestion is habitually delayed, the cause is to be sought for in defective secretion, impaired propulsion, or a combination of the two. The difficulties and uncertainties of the former have been already briefly touched upon, and propulsion comes next in order of consideration.

Impaired propulsion, if more than temporary or occasional, must be followed by dilatation, unless compensation is speedily brought about through an increase in the power and thickness of the muscular coat. If through such increase propulsion is maintained, the stomach empties itself properly; and under these circumstances the cases must be rare in which we can determine with certainty the presence of dilatation. If compensation is not established or fails, food is retained, and through its weight and decomposition tends constantly to increase the size of the organ. This brings us to a division of the subject toward our knowledge of which the sound has rendered services not to be denied even by those of the most sceptical temper. As long as we were forced to rely on palpation and percussion, even with the additional aid derived from the development of gas in the stomach, it was impossible to arrive at an accurate knowledge of the size of the stomach in all cases. At one time I attached considerable importance to auscultatory percussion; but longer experience convinced me that, in my hands at least, it is not to be depended upon. Blowing up the stomach through the sound by a Davidson syringe, however, seems to me to yield positive results, and is certainly easy alike for the patient and physician; and yet we must remember there is normally a considerable variation in the size of the stomachs of different people; that of two stomachs of the same size one may be dilated, the other not. We are probably safe in assuming that a stomach, the greater curvature of which reaches below the navel, is dilated; but we must also bear in mind the fact that unusually vertical positions of the organ are encountered, not necessarily associated with dilatation. However, it still remains when all is said that we are now in a position to detect with ease and certainty all

notable degrees of dilatation. If the use of the sound had taught nothing else than this, we could still be duly grateful.

The salol test for the motor sufficiency of the stomach, as proposed by Ewald, has been shown by recent experimenters to be unreliable; and its modification, the length of time required for the entire elimination of the salicylic acid, as proposed by Huber, cannot be said to be very practical, even if it is free from material sources of error.

The potassic iodide test for the rapidity of absorption, if carefully carried out, seems to be open to less criticism.

It appears to me that the state of mind of one who has watched the evolution of these modern methods, must be at present one of reserve. As one tries to look at the matter broadly the changes in the methods themselves, as well as in the interpretation which is to be put upon their results, have been so rapid, and oftentimes so radical, that one cannot be blamed for believing that the end is not yet. The old saying, that the stomach is not a test-tube, is emphasized. Indeed, physiological considerations should prepare us for great difficulty in drawing conclusions from the minute physical examination of the stomach. The heart and lungs act rhythmically—now under less, now under greater pressure, it is true, but continually and within comparatively narrow limits of variation. Their functions are relatively uniform. It has, therefore, been possible to build up a system of physical diagnosis which enables us to ascertain with, on the whole, wonderful accuracy the physical condition of those organs in health and disease. The kidneys, again, act continuously, pouring out a secretion which varies notably with temperature, exercise, diet, and the period of digestion; but varies, after all, within reasonable limits. Still, if we look at urinology from the historical point of view, we are impressed by the fact that the attainment of the measure of knowledge which we possess to-day has involved years of work by numbers of unwearied observers.

When I began to study medicine, twenty odd years ago, I was taught that albumin in the urine was of probably, casts of almost surely, fatal import. If accurate knowledge has been so slow to crystallize about thoracic exploration and urinary examination, can we expect in ten years to attain corresponding accuracy in the application of physical diagnosis to an organ presenting far greater difficulties in some important respects than the heart, lungs, and kidneys? There is no

organ in the body, save only the brain, perhaps, which varies so widely as the stomach in physiological activity. To take an extreme case: Contrast the stomach of Dr. Tanner, late in his fast, and that of a Hottentot successful in the chase. In any person, within a period of twenty-four hours, the stomach varies between full work and absolute rest, besides undergoing notable changes in size. The wider the limit of physiological variation the harder to separate physiology from pathology. Now, I submit that there is a close correspondence between the difficulties which have been encountered and the uncertainties which still confront us in the practical application of the results of the modern methods to gastric physical diagnosis, and those which physiology and analogy would lead us to expect. Only two years ago I heard two justly very eminent members of the profession proclaim in public their conviction that these methods were about to put us in the diagnosis of gastric disease where we are in the diagnosis of renal disease. I trust that I am neither very ignorant nor very prejudiced in this matter, but it is my belief that our main reliance in gastric diagnosis must still be placed upon the rational and the coarser old-fashioned physical signs. I should be sorry to convey the impresion that in my poor opinion the labors of many devoted men are valueless; on the contrary, I believe that at least a foundation has been laid, and walls are rising. It may be necessary to pull down and reconstruct here and there for a time, but the building will be completed. All I maintain is, that it is far from being ready for occupancy, save in a corner here and there. I should sum up my estimate of diagnostic results somewhat as follows:

1. The sound renders it possible for us to determine accurately the length of time required for digestion.

2. It also enables us to determine with far greater certainty than was formerly possible, whether the size and seat of the stomach have undergone any notable pathological alteration.

3. It is contributing toward the classification on a pathological basis of the conditions hitherto embraced under the vague clinical term "dyspepsia."

4. The chemical study of the gastric contents, when carefully and persistently carried out, is an aid in the diagnosis of some cases, but its value is still quite subordinate to that of the rational signs.

5. Finally, the chemical methods themselves are so time-consuming,

they must be so frequently repeated, and their results are so uncertain, that in their present form their application presents almost insuperable difficulties, except to the hospital physician, who must also have a larger staff of trained assistants than is usual in American hospitals.

DISCUSSION.

DR. G. L. PEABODY: Although I have but very little to add to what has been said I have, nevertheless, to say that I am firmly persuaded of the very great value of these exact methods in enabling us to make diagnoses, which without them we would be unable to reach with any certainty. It seems to me that Dr. Kinnicutt has not overstated the merits of these recent methods when he says that the continuous and persistent presence of free hydrochloric acid in the gastric contents is enough to justify us in excluding the diagnosis of cancer. A case in point is at the present time under my observation. He has been seen by Dr. Kinnicutt with me. The very great emaciation which has resulted from his protracted illness, and the fact that he is beyond middle life, would have led me to believe some years ago that he had cancer. But from the fact that there is always present in the stomach-washings, as well as in the vomited matter, free hydrochloric acid, I have diagnosed the case as one of benign stenosis of the pylorus, and have felt justified in recommending the man to submit to a surgical operation, as I believe that his ultimate fate could at least be postponed by this method of procedure.

The methods, however, are certainly difficult of application by the busy practitioner in any but a well-regulated hospital, with laboratory attachment and a corps of assistants. I think Dr. Kinnicutt would be willing to admit that without assistants and without hospital facilities it would not be practicable to apply these methods in all cases. I think, however, we ought to be grateful for the light they have given us, and, even though we have not, as Dr. Shattuck says, yet reached positive conclusions as to their value in all directions, we ought to be grateful to those who have done work enough in this direction, either original or corroborative, grateful to them for showing us in some of those cases which are so difficult of diagnosis that there is at least a ray of light thrown by these chemical manipulations.

DR. F. FORCHHEIMER: While in the main I agree to what the previous speaker has said, I would like to emphasize one point. There are some things which struck me, from the standpoint of the general practitioner, as being necessary to be more particularly brought out. I refer especially to the difficulty with which all these examinations are carried out. To those of us in active, busy life who have to see a great number of patients, it seems almost impossible to carry out these methods of investigation. Dr. Kinnicutt made

mention of one test requiring fifteen minutes. In general practice we will have two or three of these cases under our observation at the same time; and, in an individual case, in order to come to conclusions, it is necessary to make not only examinations in one direction, but several. It thus becomes absolutely impossible to make these examinations in general practice. Therefore, as far as we have gone, it seems that it is only feasible to carry them out in hospitals, and in hospitals exceedingly well appointed and having in connection with them physiological or pathological laboratories.

The second point was, that in order to come to conclusions by means of examining the contents of the stomach, it is necessary to make a complete method of it. In other words, it is not sufficient in making the examination to determine the degree of acid or the quantity of hydrochloric acid or of any single constituent; it is necessary to go through all the tests, making it much more difficult, even, than those who are willing to carry out the tests in a rudimentary way care to undertake.

The method of Ewald includes the examination of the starches, and I am glad to see Dr. Kinnicutt has dropped this. It is not at all necessary to make the various tests from starch to dextrose.

The digestion of starch in the stomach is of comparatively little importance. The prevention of the continuance of the salivary function in the stomach does not throw new light upon any pathological condition of the stomach, as a cause for the latter would have to be found. This is done by carrying out the method of analysis given by the essayist.

One other point, and that is as to the unreliability of the test of one substance formed from starch and dextrose, viz., achroö-dextrine. I have had them take place in a simple solution of starch where I was positive with other tests that such a substance would prove to be not present at all.

I think with Dr. Shattuck that at present we are on the threshold, and, so far as the general practitioner is concerned, we are a little further than the threshold; but, so far as the general practitioner is concerned, these methods cannot be utilized at the present time.

Dr. J. West Roosevelt: In speaking of these tests it seems to me one must not consider the general practitioner as necessarily the man who is to make these tests, any more than one considers the general practitioner the person to examine pathological specimens. It would seem if a chemical examination of the fluids of the body, of all the various organs, is to be undertaken, it must be by specially trained men. It strikes me as unfortunate that objection should be made to tests on the ground that they are not easy for the general practitioner. It does not seem at all fair to consider any tests easy to an inexperienced man. It ought not to be objectionable that the tests take time, are difficult, or complicated, or anything else, simple because they are convenient to the general practitioner. It is of the highest importance that the general practitioner should learn (I personally learned it in a chemical laboratory) that chemistry is not to be undertaken in a careless, cheerful kind of spirit, with the idea that his results are going to be of value, if he is an

untrained man. Work of this kind is of the highest importance, but I do not think we should begin using any of the methods with the idea that they are easy for anyone who has not made the tests many times.

DR. KINNICUTT: I cannot agree with Dr. Forchheimer that these methods of examination will prove difficult to the general practitioner. I am confident that if any intelligent physician will give a few hours to learning the technique of the methods and tests which have been described, he will be convinced of the simplicity of their application. I have found no difficulty in making use of these methods in my office consultations. Moreover, I am firmly convinced of their great practical value, and I earnestly trust that their more general employment may not be neglected.

NOTES OF TWO CASES OF ACROMEGALY.

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It has been well said that he who gives a name to a certain series of signs and symptoms not previously classified confers a benefit on science.

In 1884 Fritsche and Klebs published a long article entitled "Ein Beitrag zur Pathologie des Riesenwuchses," in which an exhaustive description of a case is given, together with a list of similar cases published under various names.

It was not, however, until after 1886, when Marie published his first paper and introduced the term acromegaly, that the attention of the profession generally was drawn to the study of this disease. Since that time a number of cases have been reported and some progress has been made in its etiology and pathology. I might here refer to the exhaustive article of Erb, published in the *Deutsches Archiv für klin. Med.*, 1889.

In the July number of *Brain* Marie gives a summary of cases published up to that time. In this list appear eighteen undoubted cases, four probable ones, and six which, although published as acromegaly, Marie did not consider as examples of that disease. Two cases reported on this continent were not included in this list

A case has also been published by Drs. Holschewnikoff and von Recklinghausen, in the last January number (1890) of Virchow's *Archiv.*

Dr. Henry Waldo, of the Bristol Royal Infirmary, reported a case which appears in the *British Medical Journal* of March 22d of this year. This was of an acute form, lasting but six months, and on post-mortem examination it presented some gross brain lesions, which were

very interesting and which will be referred to further on. The shortness of duration of this case, the absence of marked head and face enlargement, the normal size of the inferior maxilla, and the peculiar condition of the skin, all make it doubtful whether this ought to be included in the list of genuine cases, or whether it is not a trophic neurosis which Virchow would describe as a partial acromegaly.

Within the last day or two my attention has been drawn to a letter in the last number of the *Medical World*, written by a physician in the State of Missouri, in which the description of a patient is given who is probably suffering from acromegaly.

Of the cases published on this continent, one by Dr. Wadsworth appeared in the *Boston Medical and Surgical Journal* of January 1, 1885, under the heading "A Case of Myxœdema, with Atrophy of the Optic Nerves." Marie includes this in his list of genuine examples. To my mind the case presented many symptoms of myxœdema as well as those of acromegaly. Changes in the skin and subcutaneous tissues were described such as are found in cases of myxœdema, while the enlargement of the bones would place it under the head of acromegaly.

The second case was reported by Dr. Adler, before the New York County Medical Society, and published in the *Boston Medical and Surgical Journal* for January, 1888. This appears to have been a good example of the disease as it has been described by Marie. The striking peculiarity of the case was a widespread enlargement of the lymphatic glands. There were no distinctive signs of myxœdema present.

The third case was published by Dr. O'Connor in the *American Journal of Homœopathy*, 1888. It is also a genuine case of the affection; "an extreme infiltration of the lining membrane of the cheeks and soft palate" is described. Whether this resulted from a simple hyperplasia of the connective tissue, or from an infiltration of mucin, is not clear—possibly the latter, as the doctor thought "some purely myxœdematous symptoms were undoubtedly present."

It would thus appear that three cases have already been reported on this continent. So far as I know, the two now to be described are the fourth and fifth in the order of publication.

In August of last year Dr. Osler, who was then on a visit to Toronto, drew my attention to a case in the Toronto General Hospital,

under the care of my colleague, Dr. Burritt, which he (Dr. Osler) considered to be a case of acromegaly. The patient was afterward transferred to me.

I am, therefore, indebted to Dr. Osler, who made the first diagnosis, and to Dr. Burritt, for some of the earlier notes of the first case.

The second patient I had frequently seen and put him down at once as an example of acromegaly after the first had been shown me.

The following notes of the first case were made partly in August of 1889, but principally in April of this year.

CASE I.—G. B., æt. forty. Born in Canada. When young he suffered from severe attacks of asthma, which ceased when he was about seventeen years of age. When twenty, he suffered from an attack of bronchitis, which was probably complicated by asthma. This lasted about a year, and his physician was apprehensive of tuberculosis. He, however, recovered, and has not since been troubled with asthma. At twenty-two years of age he suffered from malaria, which continued six months. He then lived in a miasmatic region.

From that time up to the commencement of the present illness he enjoyed good health. He was a strong, well-built, athletic man. He thinks he worked too hard on the farm. At thirty-five he married. His wife died three years ago. One child was born, who died in infancy of some intestinal trouble.

Family history.—His father, a native of England, died aged seventy-five. For years previous to his death he suffered from chronic rheumatic arthritis. His mother died at sixty-five, of an obscure disease of the bowels. He has eight brothers and two sisters, all living and healthy. He knows of no case similar to his own in the family.

When about thirty-five years of age he complained of severe pains in the left side of his face, which were thought to be of neuralgic character. He had several teeth extracted, without, however, giving him any relief. Some months after this he noticed a prominence of the left malar bone and an enlargement of the left ramus of the inferior maxilla. The presence of the malar enlargement was brought to his attention by a physician whom he saw occasionally, and who told him he had a tumor forming on the left side of his face.

About three years ago, two years after the commencement of the neuralgic pains, he noticed a general enlargement of the face, and at the same time of the hands and feet. The pains have continued without intermission for the last five years. They are of a dull, aching character, and are often accompanied by dizziness and a peculiar indescribable feeling of distress in the head.

Three years ago he noticed his sight failing, first in the left, then in the right eye. This diminution of sight has steadily progressed up to the present.

He has suffered much from chronic constipation, and from hæmorrhoids which frequently bleed. His appetite has always been good. He has been in the habit of drinking large quantities of water, and has taken spirits moderately.

He has at times noticed polyuria, which would last for a month or so. Of late weakness and shortness of breath resulted from moderate exertion.

At the commencement of this illness he weighed 180 pounds. He now weighs 212 pounds, and is about five feet nine inches in height.

Present condition.—Patient is pale and sallow. The face, hands, and feet are noticeably larger in proportion than the rest of his body. He walks slowly and heavily, with his shoulders stooped, owing to a rounded curvature of the spine. His face is much enlarged, and elliptical in shape. The skin over the face, of a sallow color, is loose and easily movable. It has a soft feel, and can be pinched up in large folds. There is no appearance anywhere of a deposit of mucin such as occurs in myxœdema. Length of the face from the top of the forehead to the tip of the chin 245 millimetres. Breadth between the malar prominence 160 millimetres. The forehead is low and receding. The supra-orbital arches are very large, especially the left one. The prominence of the eyebrows gives the eyes a somewhat sunken appearance. The malar bones are much enlarged, the left one being more prominent than the right. The nose is very voluminous, but is not pugged. It is uniformly enlarged. The upper lip is elongated and somewhat thickened. The superior maxillæ are enlarged at the upper part. The alveolar surface, as is shown in the model, is about normal in size, except at the posterior part of the left side.

The inferior maxilla is enlarged in every way, and is set forward so that there is about a third of an inch between the teeth above and below when the mouth is closed. He says before the disease began he could bite a pin in two. He has had some teeth extracted, and others are decayed. The tongue is enlarged, and presents a red, furrowed appearance. The raphe is deep, and deep furrows branch out on each side.

The enlargement and position of the inferior maxilla give to the lower part of the face a heavy appearance. The lower lip is long, thick, and protruding. The chin is long and heavy. The hair in the moustache is thin and not strong. His beard does not grow nearly so fast or so strong as before the commencement of his illness.

The ears are uniformly enlarged.

The cranium does not appear much larger than normal.

The neck is not much increased in circumference. It measures 410 millimetres.

The right lobe of the thyroid can be felt with great difficulty, but no other part of the gland.

There is a slight curvature of the spine forward in the lower cervical and upper dorsal regions, and a corresponding lordosis in the lumbar region.

The hands are broad, flat, and fleshy. On grasping the hand it feels soft,

and so large that it cannot be properly held. The right is slightly larger than the left. Part of the index finger of the right hand was lost in an accident when he was three years old. The metacarpal bones are especially lengthened. The carpal joints are much enlarged and flattened, especially those of the index and middle fingers. At the side of the metacarpal bone of the little finger there is a mass of soft tissue, but not out of proportion to the fleshy condition of the rest of the hand. The palmar lines are deep and well marked. The skin presents a yellowish appearance, and is loosely

attached to the subjacent tissue. The nails are short, broad, and presen transverse markings; some of them are curved, so as to be sunken in the centre and turned up at the edges. The forearm is normal in size, measuring in circumference midway between the wrist and elbow, right 280 mm., left 260 mm. The elbow is somewhat larger in proportion; circumference over the head of the radius when the arm is straight, right 300 mm., left 287 mm. The arm midway between the elbow and shoulder joints measures, right 315 mm., left 300 mm. The scapulæ are not enlarged.

The feet are also increased in size; he has now to wear boots three sizes larger than five years ago. The feet are elongated and fleshy, but not particularly flat; there is no large mass of flesh found at the side of the metatarsal bone of the little toe, as was found in Marie's cases.

The foot measured antero-posteriorly, right 257 mm., left 269 mm. Circumference of the heel and instep, right 370 mm., left 385 mm. Greatest circumference of the foot, right 310 mm., left 290 mm. From these measurements it will be seen that the foot is uniformly enlarged.

The toe-nails are short and broad.

The ankle measured, right 270 mm., left 260 mm. The leg measured midway between the ankle and knee, right 365 mm., left 352 mm.

The knee-joints are slightly enlarged. Both patellæ enlarged. The thigh midway between the knee- and hip-joints measured, right 535 mm., left 530 mm.

The trunk does not present such abnormal conditions as will be described in my second case.

The clavicles are much thickened, especially toward the sternal ends; the scapulæ are not enlarged, and the sternum is not prominent; marked dulness was found over the upper third of the sternum.

The chest measures over the nipples 1020 mm.; the abdomen over the umbilicus 930 mm.

His respirations are diaphragmatic in character.

April 27, 1890. His pulse 78; temperature normal; respiration 17. His heart is normal in size; there is an accentuation of the second sound. The lungs are normal. There is no enlargement of the liver and spleen.

About a year ago he passed large quantities of urine; he thinks it is now normal in quantity. The following analysis was made by Dr. Caven: Reaction strongly acid. Color normal. Sp. gr. 1021. Albumin about a seventh by measure. Sugar none. Pus cells in considerable number. Abundance of oxalate of lime crystals. No casts.

There is a chain of indurated and slightly enlarged lymphatic glands along the posterior margin of the sterno-mastoid muscle on each side. A similar condition is found in some of the glands in the posterior part of the neck. There is no evidence of any other changes in the lymphatic system.

The organs of generation are normal in size. For the last three years he has had very little sexual feeling. A double varicocele is present.

He has a deep bass voice, which he says does not at all resemble that of five years ago. He could then sing and whistle with ease, now he cannot call out loud, and can sing and whistle with difficulty.

The patient is much troubled by a thick mucus which collects in the back part of the mouth. There is much thickening of the mucous membrane of the posterior nares; this, in all probability, is the predisposing cause of the occasional deafness from which he suffers. His appetite is always good, and he is not often troubled by indigestion.

The tendon and skin reflexes normal. Muscular power of the arms not

much diminished. Last fall he had more than the ordinary strength of muscles of the forearm; at the recent examination his grasp was not so strong.

He complains of hyperæsthesia over the left side of the neck and head, also over the gum on the left side below. There are no abnormal sensations in the extremities. He has a constant dull aching pain in the forehead, face, and teeth, from which he says he is never free.

The following account of the condition of his eyes was sent me by Dr. Reeve: Vision very imperfect, right eye, V. $= \frac{8}{cc}$: left eye, $\frac{10}{cc}$: and reads 14 Jaeger. Double temporal hemianopsia. Color-sense defective for green. Pupils of normal size and respond well to light and accommodation, but do not dilate well. Right pupil almost if not quite unaffected by pencil of light from temporal side; left pupil partially so. Gray atrophy of optic nerves, not post-neuritic. Retinal arteries not more than one-half normal size, and veins small. Divergent strabismus. No present diplopia.

His hearing is at present fairly good; taste and smell normal.

He is not intellectually bright, and says he cannot do business as well as formerly.

He is of opinion that he has rather gained during the winter months, but his appearance is not nearly so healthy as when he was last in Toronto.

Measurements (after Marie's case in *Brain*).	Right. *mm.*	Left. *mm.*
(1) Length of the hand from the lower fold of the wrist to the end of the middle finger	215	215
(2) Length of the middle finger, starting from the palmar fold at its base	95	93
(3) Length of the middle finger on the dorsal aspect, starting from the base of its first phalanx	125	123
(4) Length of the little finger, palmar aspect	73	70
(5) Circumference of the hand, without the thumb, at the head of the metacarpal bone	265	250
6 Width	113	110
7 Greatest circumference of the obstetric hand	300	285
(8) Greatest thickness of the hand at the level of the thenar eminence	55	55
(9) Greatest circumference of the middle finger	95	95
(10) Circumference of the thumb	90	85
(11) Greatest circumference of the little finger	80	75
(12) Circumference of the wrist immediately below the extremities of the ulna and radius	205	210
(13) Circumference of the wrist at the level of the styloid process of the ulna	200	205
(14) Circumference of the forearm at the middle	280	260
(15) Circumference of the arm at the middle	315	300
16 Length of the nail of middle finger	18	19

Measurements (after Marie's case in *Brain*).	Right.	Left.
	mm.	*mm.*
17 Length of the nail of thumb	20	18
(18) Breadth of the nail of middle finger	20	21
(19) Breadth of the nail of thumb	26	24
(20) Length from the iliac crest to the summit of head of fibula	530	530
(21) Length from the summit of the head of the fibula to the top of the external malleolus	390	390
(22) Vertical measurement of the patella	65	
(23) Transverse " " "	70	
(24) Circumference of thigh at the middle	535	530
(25) Greatest circumference of the calf	365	352
(26) Circumference inmediately above the tip of the internal malleolus	270	260
(27) Greatest length of foot	257	269
(28) Circumference of the heel and instep	370	385
(29) Greatest circumference of the foot	310	290
(30) Greatest width of the foot	110	110
(31) Circumference of the great toe	102	102
(32) Circumference of the little toe	65	63
(33) Length of the nail of great toe	20	26
(34) Width of the nail of great toe	28	28
(35) Length from top of forehead to tip of chin (with callipers)	*mm.* 245	
(36) Length from top of forehead to upper part of nasal bones	95	
(37) Length from upper part of nasal bones to tip of nose	70	
(38) Greatest width of alæ nasi	45	
39 Distance from tip of nose to point of junction of latter with the upper lip	30	
(40) Length from the septum of the nose to the point of the chin	105	
41 Greatest difference between outer surfaces of the cheek bones	160	
42 Width of mouth	65	
43 Vertical measurement of lower lip	18	
44 Transverse measurement of tongue at the middle	80	
45 Thickness of tongue	170	
47 Lower jaw, vertical measurement from the free border of the gums to the lower part of the symphysis (with callipers)	50	
(48) Distance (with callipers) from the temporo-malar articulation to the lower part of the symphysis of chin	180	

Measurements (after Marie's case in *Brain*).

		mm.
(49)	Distance between the two angles of the lower jaw (with callipers)	135
(50)	Ditto with tape measure along the body of the bone passing in front of the symphysis	270
(51)	Ears (R. and L.), greatest length	80
52	Ears " " " breadth	45
53	Circumference of thorax over nipples	1020
54	Circumference of neck between hyoid bone and upper part of thyroid cartilage	410
55	Circumference of abdomen on a line with ensiform cartilage	1005
56	Ditto over umbilicus	930
57	Ditto over crest of ilium	930
58	Circumference of the head over the eyebrows	620

The history of my second case was obtained partly from the attending physician, Dr. Leslie, and partly from the relatives of the patient. Although I had frequently seen him, I was never called to visit him professionally. He was sensitive about his condition, and did not like to have it referred to.

CASE II.—Mr. T., aged fifty-seven years, bank clerk, born in Edinburgh. Came to Canada when he was twenty-three years of age; nothing was at that time noticeable about his figure. He was a strong, well-built, large-boned man, about five feet ten inches in height. Shortly after his arrival on this continent the enlargement of his head and face commenced, and at twenty-eight years of age the change was so marked that his mother, whom he then visited in Scotland, scarcely recognized him, and expressed great astonishment at his altered appearance.

So far as can be recollected, the enlargement of the hands and feet began at the same time as that of the head. A gradual increase in the size of the extremities went on for years. Of late, the change in the form and size of the trunk was more noticeable.

During the last five years of his life he did not do any work. He had ample means, and seemed disinclined to enter into business. He was morbidly sensitive about his appearance, and did not care to talk about it, even to his physician. He was of an amiable, affectionate disposition, and spent most of his time at his own home, enjoying the company of his family. He was unusually bright and cheerful, fond of reading, and he talked with much intelligence.

The changes in the various extremities may be described as follows: His hands were much enlarged, broad, and flat. The carpal joints were enlarged, so that he could not slip an ordinary finger-ring over them. He wore on his little finger a signet ring, which was made to order for him. The flatness

and broadness of the hand were especially noticeable when he placed it with the palm downward on the table. A soft enlargement by the side of the fifth metacarpal bone could then be more especially seen.

His nails were short, broad, and small compared with the size of the fingers. He kept his nails closely cut, a circumstance which gave them a still shorter appearance.

The right hand was considerably larger than the left.

His wrist was enlarged, measuring 9 inches in circumference.

There was nothing noticeable about the forearm and arm, except that during the latter years of his life they became much emaciated.

The feet were also enlarged but not unshapely. The toes were elongated and flattened. (The sole of his slipper measured 12 inches in length by 4 in breadth.)

There was nothing noticeable about his legs and thighs, except a considerable enlargement of his knee-joints.

His head was much enlarged. Circumference, as shown by his hat-band, was 24 inches. His hat measured 8½ inches antero-posterior diameter by 6½ transversely.

The face was elliptical in shape. The forehead was low and sloping upward and backward. His son, who is now twenty years of age, has a similarly shaped forehead. The eyebrows were prominent, and the skin over the forehead much wrinkled.

His nose was uniformly enlarged and slightly pugged.

His lips were thickened and enlarged, the lower being much more prominent than the upper.

The upper and lower jaws were much enlarged. He had a long, fat chin, which at ordinary times rested on his chest, so that his necktie could not be seen.

His neck, short and thick, seemed to be set on the upper and anterior part of the trunk. It was much arched behind and was marked by large, thick folds of integument, which were separated by deep furrows. His ears were also much enlarged.

His shoulders were much increased in size, one more forward than the other. There was a marked curvature of the spine in the upper dorsal region. The distance along the spine from the collar of the coat to a line drawn between the upper border of the axillæ, a measurement which tailors make, was 15 inches. The usual length of such a measurement is 7 or 8 inches.

The sternum was very prominent toward the upper extremity, and the ribs were flattened on each side. The circumference of the chest over the nipples measured 47½ inches.

Besides the kyphosis, there were lateral curvature and lumbar lordosis. The latter was very marked,

He at one time complained a good deal of toothache, and eleven years before his death had all his teeth removed.

His sight was good up to the day before his death, when he become almost totally blind. His hearing was acute, as were also his taste and smell.

He had a deep bass voice which was slightly husky. He could never call out loudly, but always whistled when he wished to attract the attention of one at a distance. About the beginning of the year 1889 he caught cold, from which he was some time in recovering.

During the following winter months his family noticed that he slept more than usual. He would come down to breakfast, after a long night's sleep, looking pale and worn, and would fall asleep in his chair two or three times during the day. This tendency to somnolency increased during the early part of the summer.

The first time he consulted Dr. Leslie was about five years before his death. He then wished to obtain a certificate to exempt him from jury duty. He stated that he was nervous, and that, when repeatedly questioned, he could not hear or speak well.

He called afterward occasionally to consult the doctor about severe headaches from which he suffered. They were of the nature of migraine and often lasted one or two days. He could not take much exercise as he became easily tired. His appetite was always good, and he complained frequently of thirst. His urine was examined in January, 1889, and was found to be free from albumin or sugar.

On January 29th he called on the doctor complaining of pain in the calf of his right leg. He had been on his feet more than usual and he was troubled with varicose veins. The pain was thought to be due to phlebitis. The limb afterward became red and swollen. An abscess formed, which was lanced on July 11th. After a free discharge of matter, the patient improved until the 14th, when he became delirious. He afterward sank rapidly, and died on the 16th; apparently from acute septicæmia.

Measurements:

Circumference of chest over the nipple, 1162 mm.

Circumference immediately above the crest of the ilium, 1062 mm.

Distance from the collar of the coat to a line drawn across the upper margin of the axillæ, 387 mm.

Circumference of neck, 462 mm.

Circumference of wrist, 231 mm.

Circumference of head, 612 mm.

Right hand circumference, at metacarpo-phalangeal joints, 216 mm.

Length of index finger, 127 mm.

Length of ring finger, 136 mm.

Length of little finger, 107 mm.

Length of thumb, 105 mm.

Length of foot, 301 mm.

Breadth of sole, 110 mm.

Circumference of foot, 262 mm.

These measurements are not quite accurate, as they were made from the patient's clothing.

In the cases just described a remarkable similarity of signs and symptoms was present.

The differences will first be noted:

In the first case there was loss of eyesight from optic neuritis, a condition not present in the second. The latter patient appeared to be in a more advanced stage of the disease, as shown by the changes in the spine and thorax—changes which became more pronounced in the last five years—and which were only present to a limited degree in the first patient.

The disease began at an earlier age in the second case and lasted twenty-three years, whereas in the first it began at thirty-five, and from present appearances he will not live as long.

Albumin was found in the urine of the first and not in the second case.

In both cases there was a marked increase in size of both face and extremities, and this enlargement was due not only to involvement of the soft parts alone, but of that of the bony skeleton as well.

In neither case was there any myxœdematous condition of the skin or subcutaneous tissue.

The face in both cases was elliptical in shape, the cheek bones were prominent, the superior and inferior maxillæ were enlarged, the lips were thickened—the lower one more than the upper. The upper alveolar process in the second patient was much more enlarged than in the first.

In both patients the hands were increased in size and there was marked enlargement of the carpal joints. There was enlargement and lengthening of many of the bones and an increase of the connective tissue as well. The same description will apply to the feet.

In both cases there was a gradual lessening of vitality, although the mental faculties remained, to a great extent, unimpaired. In the second patient this diminution of vital force was more marked during the last six months of his life.

Both patients suffered from severe aching pains about the face and head.

The first patient was a plain farmer of fair education, whereas the second was a well-educated man.

There can be little doubt that both cases belong to that class de-

scribed by Marie under the title of acromegaly. That they were not cases of myxœdema is evident.

1. The dimensions of the bony skeleton were in many parts much increased, whereas in myxœdema the soft parts are alone affected.

2. The face in myxœdema presents a round, full-moon appearance. In both these cases the face was elliptical.

3. In myxœdema there is a distinct waxy-like condition of the skin and subcutaneous cellular tissue. There is also a thickened and scaly condition of the integument. In neither of the cases described was there any thickening of the skin, and only in the first was there any discoloration.

4. In neither case was the mental condition present which is so marked in myxœdema. The first patient is constantly engaged selling horses, a business which cannot be carried on without a good deal of shrewdness.

The osteitis deformans of Paget is excluded, as in it the head is enlarged and the face retains almost its normal size. In the cases described the enlargement existed principally in the face.

The deformities of the long bones which exist in Paget's disease were not found in these cases.

Osteitis deformans begins after the age of forty, while in the histories narrated the disease began earlier in life.

There was more symmetry in the enlargement of the bones in the cases described than there is in Paget's disease.

In gigantism we have a condition in which the extremities are enlarged only in proportion to the general stature. The face is not elongated and the lower jaw is not hypertrophied, or set forward, as has been described.

The leontiasis ossea of Virchow is excluded, as in these cases there were no well-defined bony tumors, but there was hypertrophy of the hands and feet—a condition not found in the disease described by Virchow.

Marie, I think, properly excludes localized hypertrophies, such as that of one extremity, or unilateral hypertrophy of the face, from cases of this affection.

It is possible that these dystrophies may be produced by localized lesions of trophic centres similar to the general lesion which may hereafter be found in acromegaly; such, for instance, as has been found

in von Recklinghausen's case. In the case just described, as well as in the twenty-two already reported, there is a distinct clinical history and distinct pathological conditions which separate them from all previously described diseases.

Although, in my opinion, there is little doubt of the existence of acromegaly as a distinct disease, it seems, in some cases, to have a close relationship to myxœdema. . For instance, in Wadsworth's case, which is put in the list of genuine examples by Marie, there was a waxy condition of the skin, as well as an enlargement of many of the bones of the skeleton. The same condition is described in Tresilian's case. From this it would appear that although in most cases of acromegaly the skin simply undergoes the same hypertrophy as the subjacent soft parts, there are those in which a myxœdematous condition is found in addition to the hypertrophy of bones and soft parts.

In studying the possible connection between these two affections two sources of error must be guarded against. Cases may have been described in which the skin and subjacent tissues were myxœdematous, when the waxy condition was not really due to a deposit of mucin. Again, as Virchow suggests, in cases of genuine myxœdema the bones may be enlarged to a considerable extent without its being noticed, on account of the thick covering of integument and connective tissue infiltrated by mucin.

On account of the small number of post-mortem examinations made, there is much obscurity as to the true nature of this affection. I regret very much that in this respect the report of my second case is so defective.

In one of Marie's cases Broca has described the changes in the skeleton as follows: "The spongy tissue is especially the seat of hypertrophic changes, so that the following statement may be considered as representing the reality: In the skeleton of acromegaly hypertrophy shows itself in the bones of the extremities and in the extremities of the bones."

With regard to the morbid anatomy, Marie comes to the following conclusions: "Until proof to the contrary is brought forward, I shall cling to the belief that these three anatomo-pathological characters manifest themselves not only with a remarkable degree of frequency, but may even be looked upon as constant, viz., hypertrophy of the pituitary body with enormous dilatation of the sella turcica, persistence

of the thymus, and hypertrophy of the cord and ganglia of the sympathetic system."

Klebs mentions prominently the enlargement of both veins and arteries, and particularly the increase in size of the valves in the veins. In some cases the thyroid gland has been either atrophied or diseased, whereas in others it has been found normal.

Since the publication of Marie's paper, in which the foregoing statement appeared, the few post-mortems made have been on cases of doubtful diagnosis. The case described by Holschewnikoff and von Recklinghausen, previously referred to, presents some points of great interest. The patient died shortly after admission into the hospital, and the clinical history is, in consequence, defective. The post-mortem was made previous to 1886, when Marie published his first paper.

The measurements, with the exception of one hand, were not made so much in detail, nor the appearance of the head and face elaborately given. The case is described as one of "syringo-myelia and peculiar degeneration of the peripheral nerves, combined with trophic disturbance (acromegaly)." It presented the peripheral and central lesions of syringo-myelia, together with the peculiar enlargement of the bones found in acromegaly.

The study of this case would direct one's attention to the spinal cord as the probable seat of the primary lesion in acromegaly. Unfortunately, as von Recklinghausen states, the spinal cord has not been carefully examined in any of the post-mortems so far reported, and until such examination has been made much doubt will surround the pathology of this affection.

In the case reported by Dr. Waldo, and already referred to, the patient was fifty-four years of age, and the disease had lasted six months. The post-mortem examination revealed cavities of the brain, the probable result of embolism. There was also valvular disease of the heart. The condition of the spinal cord was not given.

In this case endocarditis, rheumatism, and embolism appeared in combination with hypertrophy of the bones and soft parts of the extremities.

From the comparatively slight opportunities the writer has had for the study of this affection, he is inclined to agree with Marie that acromegaly is a general disease presenting features sharply defined which separate it from other pathological conditions. It would be

difficult to find two cases of any chronic affection which present greater similarity of symptoms than those just described.

A point of importance in etiology might be here mentioned. Virchow, in referring to the connection said by some authorities to exist between this disease and the development of the organs of generation, gives it as his opinion that there is no evidence of any special relationship existing between them. It will be noticed on reading over the histories already published, that in the male there is frequently weakness and even impotence, and in the female a cessation of the menstrual function follows shortly after the commencement of the disease.

In the first of the cases given there was a distinct loss of sexual feeling after the second year, while in both the disease began shortly after marriage. In both instances, again, children were begotten while the disease was in progress.

In the first case no form of treatment proved of any avail. The second did not receive any special medication for the disease.

DISCUSSION.

Dr. M. Allen Starr: Inasmuch as the case alluded to by Dr. Graham as reported by Dr. O'Connor was under my care, I will say a word in regard to it. It was the case of a woman, fifty-six years of age, sent to my clinic from the Skin and Cancer Hospital, of New York. The condition presenting itself at first was one of roughened skin, with much subcutaneous thickening. The parts chiefly affected were the hands and feet. In the course of two years this woman went on steadily to the development of a very extraordinary condition of the appearance of the face and of the extremities. The face became flatter and broader, the nose, particularly, becoming prominent, and the hands and feet were enlarged. This condition was associated with a very manifest thickening and hardening of the skin, but without œdema. There seemed to be a diminution in the size of the neck. I had regarded the case as one of myxœdema, but I am convinced that Dr. O'Connor's diagnosis, which was made a year after the woman passed from under my observation, is the correct one, because, toward the close of my care of her, I noticed a perceptible increase in the size of the bones not present when first under observation. She suffered a change, also, in the tones of her voice; they became gradually hoarse, and there was a lowering of the tones. She was lethargic, slow in her replies, and manifestly stupid, more so at the close than at the beginning of the observation. These cases, it seems to me, are of considerable interest, and I think we ought to be grateful to Dr. Graham for putting on record such complete observations. Such observations add largely to our knowledge of a rare disease.

THE DIATHETIC CAUSES OF RENAL INADEQUACY.

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THE term *diathesis* is defined by Thomas as a particular habit or disposition of body. For present purposes I propose to extend the meaning of the term beyond its etymological limit, including thereunder any habit or disposition of body which tends to overtax the kidneys and thus bring about the conditions prodromic of renal inadequacy. By *renal inadequacy* I mean partial or complete suspension of the renal function, generally for brief periods only, without lesions perceptible to the naked eye—that is, without lesions recognizable by the ordinary method of conducting cadaveric sections. In fact, I deem it quite possible that fatal renal inadequacy may occur without any morbid changes capable of being recognized by the unaided eye, either in the renal vessels or tubules. At the same time I hold that there is no such thing as intrinsic renal failure, without an intrinsic material cause in the nature of a morbid change, either in the epithelia of the convoluted tubes or in the bloodvessels of the cortex; in other words, there is no such thing as functional disease without a structural lesion, however insignificant that lesion may seem to be. So long as a given cell carries on its molecular changes normally we cannot conceive of any inadequacy in its functional power, and the same law applies to all cells of all the organs of the body. I, of course, recognize such extrinsic causes of renal inadequacy as surgical shock, degenerations of the spinal cord, and other sources of like nature, but, as they do not come within the scope of this paper, I shall not allude to them again.

In the next place, I believe that, in the great majority of cases, the causes of transitory renal inadequacy are directly traceable to certain "diatheses" or "habits of body" whereof the lithic acid, the oxalic

acid, and phosphatic diatheses are most frequently encountered, and are, therefore, mainly responsible. At this point I ought to say that I do not use the term "diathesis" as indicating a fixed or definite pathological process or sequence of events, but rather as a convenient name for a pathological condition, without discussing the question of the origin of that condition. At the same time it is proper for me to add that I do not admit the force of the arguments of some writers, notably Ebstein, wherein they attempt to show that the diatheses in question are founded upon insufficient grounds and ought, therefore, to be given up. It is enough for present purposes to say that the *conditions* which, for convenience, I have called diatheses, frequently exist; that they are often marked by renal failure, more or less complete; are productive of certain well-defined symptoms; that they are accompanied by changes in the renal epithelia which, though slight, are definite and uniform, and that the ultimate result, if the disease be left to itself, is some graver and generally fatal lesion, as Bright's disease, renal calculus, or suppurating kidney. The ultimate changes in the kidney are substantially the same, whether the case be one of lithæmia, oxaluria, or phosphuria; but as the former is the most frequent, and as its effects are usually progressive and uniform, I shall speak of this at length and in detail, and then refer to the two last named only as they present special peculiarities.

In my experience, the presence of lithic acid in the urine in abnormal quantity and for any considerable time is invariably followed by failure of the renal function more or less pronounced. Many years ago this clinical fact attracted my attention, and awakened on my part a desire to investigate the changes in the renal structure which at once produced and explained such failure. I have accordingly for the past ten years tried to keep pretty close track of my cases of renal insufficiency, and have taken special care in studying the urinary sediments in such cases, making repeated observations while such cases were under treatment. In many instances the same patient has been under observation for several months, and, in not a few, for more than three years.

As already stated, I find lithæmia by far the most frequent of the three prominent urinary diatheses—at least, in Chicago. From conversations with medical men from other parts of the country, as well as from the indications of our medical journals, I am inclined to

the belief that lithæmia is far more common in Western cities than in the Eastern seaport towns, and I think this fact is quite easily explained. Our Western cities are nearer the source of animal-food supply; hence, flesh foods are cheap and easily obtained, and are largely consumed. It is possible, also, that the more general consumption of malt liquors, especially lager beer, in Western cities has something to do with the production of lithæmia, although my personal observations among our German population do not lead me to this conclusion. I am inclined rather to adopt Fothergill's conclusion that "malt liquors, when taken with excessive quantities of food, especially animal, unquestionably tend to produce gout"—that is, lithæmia.

I am accustomed to regard the renal changes induced by lithic acid as passing through three phases or stages of development, namely: *First*, the stage of "irritation," during which the epithelia of the convoluted tubules undergo a simple hypertropy without hyperplasia. I would designate it as the stage of *irritative hypertrophy*. If an individual cell be examined with a good objective of fairly high power, it is seen to be slightly enlarged, which is due to an increase of both its elements. In some instances it seems to be very slightly clouded, in others no such change is noticeable. In no case can it be regarded as a fairly established cloudy swelling; in a word, the cells seem to be simply overgrown, but the pathological factor in the case is this, that, although the renal cells have acquired increased proportions, they have not only *not* acquired increased functional power, but, on the other hand, their functional power is actually crippled and perverted. And this is demonstrated by the following facts:

1. The total daily quantity of urine is diminished—it may be but slightly; it may be so markedly that the patient notices it himself; but to some extent there is evidence of renal inadequacy.

2. The urine is more than normally acid—in many instances intensely so.

3. The urine when voided is clouded with mucus, but does not undergo the rapid ammoniacal decomposition so characteristic of cystitis.

4. An early and copious deposit of lithic acid occurs, most commonly in the form of the well-known "red-pepper" grains. This is the simplest and most innocent form of lithæmia. It is accompanied

by no well-marked or positive symptoms; in fact, it seems almost a stretch of the imagination to designate it as a morbid process at all.

Such slight attacks are constantly happening among our active, hard-working, brainy business and professional men, without the knowledge of patient or physician. They last a few days and then disappear spontaneously, leaving no apparent record behind; yet every slight lithic shower, however short may be its duration, does leave behind it a long-enduring *irritament*, to use the terse and potent term of Virchow, in the shape of hypertrophied and angered epithelial cells, which may resume their physiological duties slowly, or pass on to the next and more dangerous stage with eager alacrity. The cause of this very slight and generally transient change in the renal epithelia is undoubtedly the presence of lithic acid in the blood. There seems to be a well-pronounced and positive incompatability between lithic acid and the protoplasm of the renal cells, in so much that the presence of lithic acid is simply intolerable to the cell; in so much that the presence of lithic acid in the blood, in any appreciable amount and for any length of time, provokes such changes in the cell that it is incapable of carrying on its perfect work, and thus renal inadequacy is established, and thus we are forced to recognize a "diathesis" as a cause of renal obstruction.

The *second* stage I have called the stage of intratubular crystallization or granular deposit. This change is generally continuous with the first stage. It is simply an event in the evolution of a continuous morbid process; but the great mass of cases first seek medical aid when this stage is reached, hence, it often seems to be the initial process. At this period there is not only hypertrophy, but there is "hyperplasia," or numerical hypertrophy, of the renal epithelia as well. Moreover, very many of the renal cells are beset with crystals or granules of lithic acid, more frequently the latter. Many of the epithelial cells appear granular, as though they had entered upon the first stage of fatty degeneration. If appropriate tests be applied, however, this clouded appearance promptly disappears, and the cell is then seen to be normal, except that it is swollen and turgid. The cell also presents a peculiar glassy, shiny appearance, due to the envelope of tenacious mucus in which it is embedded. Presumably, there is a slight hyperæmia of the renal cortex, yet not sufficient to produce diapedesis or exudation, although I am bound to admit that

this opinion is largely conjecture, since the post-mortem table furnishes few opportunities for verification.

The urine is scanty (frequently not more than 12 to 24 fluidounces per diem), high colored, of high specific gravity (generally as high as 1030), and often productive of much cystic irritation on account of its intense acidity. When first voided it is frequently cloudy, suggesting the presence of a great quantity of mucus, but if a specimen be gently heated the cloudiness disappears, only to reappear when the specimen cools. Albumin is hardly ever present during this stage, but if so, only as a trace, and temporarily. The urinary sediment is well worth a careful study as an object lesson in diagnosis and pathology. The careful observer will find—

(*a*) A deposit, more or less copious, of lithic acid in crystals or granules or both.

(*b*) Epithelia from various portions of the urinary tract, but notably from the convoluted tubules, which will present some very characteristic appearances, viz.: they will be swollen, distended, turgid, rounded, and the characteristic rods of Heidenhain will be obscured. They will be surrounded by a layer of dense gelatinous mucus, which will sometimes give them a glassy or hyaline appearance, or sometimes a dull, clouded appearance. Many of them will contain crystals or granules of lithic acid, and it is important to note that these crystals do not adhere to the cell, but are imbedded in its protoplasm, which, I think, clearly demonstrates that such crystals were formed while the cell was yet in its proper place in the tubule. Many of them will also contain two or more nuclei, a fact which is very common to epithelial cells which are teased and worried by some exciting cause until they reach a catarrhal condition.

(*c*) Considerable numbers of hyperplastic epithelial cells with quite large nuclei, the result of numerical hypertrophy, or multiplication of cells, which takes place in the convoluted tubules.

(*d*) Long, sinuous, wavy, or tortuous threads of mucus, the so-called mucin casts, which are formed in the convoluted tubes from the substance secreted by the epithelial cells which I have just described. These casts must be carefully distinguished from the hyaline, fibrinous casts of Bright's disease, since they are totally different as regards diagnostic importance, pathological significance, and origin. A very positive dividing-line can be drawn between them. The mucin casts

are the product of morbid metabolism on the part of the epithelial cells, but they are never the product of an exudate from the blood-vessels. The hyaline casts are the product of a morbid exudate from the bloodvessels, but they are never the product of cyto-metabolism. These mucin casts often contain crystals of lithic acid, or lithates, and now and then an epithelial cell or mucous corpuscle, and these bodies are *within*, not simply adherent *to* the casts, showing that they were formed prior to the cast, else they could not be imbedded therein.

(*e*) The urinary sediment also generally contains a few of the so-called leucocytes, which they probably are *not*, if by leucocytes we mean the white blood-globules, since at this stage the conditions necessary for diapedesis do not exist. It is far more probable that these little bodies are the free nuclei of disintegrated epithelial cells, and that the name of mucous corpuscle would be more appropriate than leucocyte. If, during this stage, the kidney be examined, it will be found slightly hyperæmic, but not enough so to attract the attention of the ordinary observer, and such hyperæmia will be a consequence rather than a cause of the morbid condition.

The *third* stage of lithæmia I have denominated the *albuminuric* stage. It is generally, I think, but a continuation of the second stage which I have just described. In this stage there is present—sometimes only now and then, sometimes uniformly—a trace of albumin—a *slight* trace, only to be detected by careful testing. It is so small in quantity that it is frequently overlooked, or even obscured, by the too prodigal use of reagents. The urine is generally normal in specific gravity, but high colored and intensely acid, yet it presents no abnormal appearances to the eye. It is not clouded by mucus, and the amount of sediment is very small—not sufficient to suggest anything abnormal to the observer. But after standing a few hours a very copious precipitate of lithic acid or its compounds is formed, and the nature of the case is then clearly revealed. If this deposit be examined microscopically, the observer will now and then discover a very small hyaline cast, entirely structureless, or only containing from one to half a dozen leucocytes. The long, wavy mucin casts are generally present, but the copious deposit of mucus noted in the former stage is generally wanting, and this fact denotes a marked change in the type of the pathological process which is taking place in the kidney.

In the two former stages, or groups of cases, inflammatory excita-

tion was limited to the renal cells, and did not involve any essential change in the bloodvessels or of the transit of blood within them; in fact, we had to deal only with hypertrophy or hyperplasia, or both, of epithelial structures. But in the stage, or group of cases, now before us we have to recognize an inflammatory lesion of the bloodvessels, with corresponding changes in the circulation; in fact, an exudative inflammation of so mild a type that, perhaps, a majority of cases escape observation altogether, for, like the two preceding stages, it is almost without symptoms, and utterly wanting in symptoms which are either uniform, pronounced, or pathognomonic. There is usually some lumbar pain which resembles, and perhaps is lumbago.

Generally there is disturbed digestion, with acid eructations; a torpid hepatic function, and constipation, and there is uniformly well-marked cardio-vascular tension, demonstrable by the stethoscope and sphygmograph; but these phenomena are so common and so often associated with other morbid states that their differential diagnostic value is next to nothing. Yet it is infinitely important that the albuminuric stage of lithæmia and other diatheses be recognized, because it is the curable stage of Bright's disease; and, I add without hesitation, that a proper study of the urine, chemically and microscopically, will always lead to a correct diagnosis. It is probably no over-statement of fact to say that all cases of chronic interstitial nephritis, and many cases of chronic parenchymatous nephritis, pass through this diathetic stage, and that the morbid tendency is clearly recognizable and easily remediable, so long as the morbid process is limited to the phenomena and lesions I have described.

When we come to consider oxaluria, or the diathetic causes which lead to the production of oxalic acid and its compounds, we find the mystery nearly as great as when Bouchardat declared it to be "one of the most difficult problems of the physician"; but we shall probably all agree with Maclagan that "it is the nervous system which is active in the genesis of oxalates," or, in other words, that oxaluria is generally associated with some perturbation of the nerve-centres. My own experience is quite in accord with d'Estrées in relation to the coexistence of uric acid and oxalate of lime, although I am not persuaded that this is due to a cause which is common to both.

So far as the kidneys are concerned, there is this difference to note between the effects of lithæmia and oxaluria: in the latter the symp-

toms are more uniform; there are no distinct stages or epochs which are recognizable, hence there can be no distinctive groups of cases established. There is similar uniformity in the urinary sediments. We find in the tubules of the epithelia mucin casts containing the well-known octahedra of calcic oxalate, a few leucocytes, and in severe cases now and then a small hyaline cast. There are generally multitudes of crystals of calcic oxalate floating free or in little masses, beside those in the mucin casts; it is not very unusual to find a few blood-globules, especially if the crystal-containing casts are very abundant, and in such cases the urine contains a little albumin. It is almost needless to add that renal inadequacy follows as a matter of course. It may not be so marked as to attract the patient's attention, but the fact is easily demonstrable by accurate observation. The degree of renal failure generally bears a close relation to the number of mucin casts found in the urine, just as *they* bear a very close relation to the degree of catarrhal excitation of the renal epithelia. The crystals of calcic oxalate are very frequently deposited in the renal tubuli, and the kidney is strangely tolerant of their presence. They do not seem to exercise anything like the irritating effects upon the renal tissue that follows the presence of lithic acid; hence, the subjective symptoms are less pronounced, and hence, oxaluria is likely to exist frequently and continue long unrecognized. My own clinical experience warrants the conclusion that such is the fact. Oxaluria appears to be a peculiarly American disease. D'Estrées, physician to the celebrated Contrexeville Spa, states that 80 per cent. of American patients visiting the Spa have oxaluria, a proportion very much greater than I have observed here at home. It is probably not unreasonable to conclude that many of his American patients contracted oxaluria on European soil.

In using the term *phosphatic diathesis* I do not wish to be understood as asserting or believing that there is any one fixed pathological condition or sequence of events which results in an increase of the phosphatic salts in the urine; but it is well known that many pathological conditions induce copious phosphatic showers, as shown by the sudden appearance of abundant urinary sediment and rapid alkaline decomposition of the urine. As examples of such pathological conditions may be mentioned sudden and intense cerebral excitement, chronic lesions of the nervous centres, prolonged suppuration, espe-

cially if copious and exhausting, and necrosis of the vertebræ and of the long bones.

I have a very striking example of renal inadequacy occurring every now and then in a case of Pott's disease, in which, for six months past, I have "watched" the kidneys, at the request of the surgeon in charge, for the purpose of anticipating and preventing dangerous and fatal renal failure. A detailed history of the medical side of this case during the time that it has been under my observation would be an admirable illustration of the principles I have laid down in the foregoing pages. If the sediment of phosphatic urine be examined, the same morphological elements are found as I have described in connection with lithæmia and oxaluria, except that crystals or granules of lithic acid and lithates or oxalates are replaced by phosphates. The mucin casts which I have—I think, justly—made so prominent in this paper are very abundant, and are frequently so occupied by fine phosphatic granules that they resemble the granular casts of chronic Bright's disease. I have many times had specimens of urine sent to me for examination accompanied by a diagnosis of chronic Bright's disease based upon the granular casts found in phosphatic urine. It is, of course, needless for me to say to this Association that I have not in such cases mistaken the phosphatic urine of cystitis for the phosphatic urine of renal origin.

In conclusion, I desire to place on record one prophetic utterance, viz.: When the profession at large comes to understand the importance of the diatheses in question, and when these diatheses are promptly diagnosticated and properly treated in their incipient stages, we shall see and hear very much less of chronic renal affections than we do now.

THE ETIOLOGY OF PLEURITIS, ESPECIALLY IN ITS RELATION TO TUBERCULOSIS.

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The frequent occurrence of pleuritis, its apparent harmlessness in some cases, its very serious character in others, and the great variety of conditions under which it may arise, must make it always a subject of interest. Since the discoveries of Koch, and the interest in the whole subject of tuberculosis excited by those discoveries, the study of the etiology of pleuritis and the significance of its relation to tuberculosis have received a new stimulus. Many careful observations have been made and very different conclusions have been reached by observers.

If one follows the discussions of the past few years on the subject of the relation of pleuritis to tuberculosis, he must have been thoroughly impressed with the difficulties of arriving at a satisfactory conclusion. If, as is claimed by many excellent observers, almost all cases of pleuritis not traceable to the ordinarily recognized causes are of tubercular origin, then not only must much more care be given to the diagnosis and probable etiology, but to the prognosis and treatment. If a case of pleuritis, sooner or later, goes on to the development of pulmonary tuberculosis, it is easy to accept the view that the pleuritis was of tubercular origin. The most earnest advocates of the tubercular origin of pleuritis admit that it is very difficult to prove in any given case, unless there is evidence of tubercular lesions elsewhere.

Ehrlich is of the opinion that tubercle bacilli can be demonstrated in all exudations of tubercular nature, except the sero-fibrinous. Fränkel believes it very difficult to discover the bacillus of tuberculosis in the fluids effused into the thoracic cavity, and yet he is of

the opinion that the large majority of these cases are of tubercular origin.

Von Ziemssen says that even if it is not possible to show in most sero-fibrinous pleuro-exudates the microörganisms, this is no proof of their non-existence, as they may be lost in the perforation made in the pleura by the exploring needle, or they may only occur in the form of spores.

From October 1, 1889, to April 1, 1890, I had under special observation in Bellevue Hospital, with a view to careful investigation and examination of the pleural exudates and sputa, 9 cases of pleuritis, 5 of sero-fibrinous exudations and 4 of purulent. The fluids were repeatedly examined, and were taken from the lowest portion of the pleural sac as the situation most likely to contain the cell constituents, if any were present. In not a single instance were any tubercle bacilli found. Likewise, the sputa were carefully and repeatedly examined, and in only one instance were tubercle bacilli found, and that was in a case in which the exudate was purulent. This was the only evidence of tuberculosis in the case; careful physical examination failed to reveal evidences of pulmonary tuberculosis. In one case of sero-fibrinous exudation the patient was aspirated twice in October. After the second aspiration the patient's condition improved very materially until early in March, when she developed an acute febrile attack with evidences of peritonitis, and died in three weeks. The autopsy was interesting in that it showed evidences of tubercular peritonitis, a tubercular lesion in the left Fallopian tube, and a few miliary tubercles on the left pleural surface of the diaphragm. No tubercular lesions were found in the lungs. It was the *right* pleural sac from which the fluid had been drawn in October. These cases are mentioned to show the difficulties in the way of determining the origin of pleural exudates. They are not given as instances of tubercular or of non-tubercular pleurisies.

The study of the results of inoculation of the pleural exudates has been unsatisfactory, at least in so far as the production of tuberculosis is concerned. The most recent observations in this direction have been made by von Ziemssen, who concludes that although fresh pleural exudates injected into the serous cavities of dogs, and into their subcutaneous cellular tissue, have not produced true tuberculosis, they have acted injuriously, "but the infecting potentiality could not be isolated."

It is well known that tuberculous sputa and tuberculous material from the lungs can be injected into the tissues of animals and produce tuberculosis. On the other hand, the material from so-called tuberculous joints has only to a very moderate extent the power of producing tuberculosis in animals. The potentiality seems to be limited to certain tissues. A number of observers confirm these conclusions of von Ziemssen.

From a clinical standpoint some recent observations are of great interest. At a meeting of the Leeds and West Riding Medico-Chirurgical Society, January 10, 1890, Dr. Barrs read a paper on an investigation of 74 cases of pleuritis under treatment from 1880–1884 inclusive, which had apparently recovered from pleuritis, but showed at the present time a death-rate of 57 per cent. Excluding the empyema of children, he thinks the majority of cases of pleuritic effusion are of tubercular origin. Such a series of cases lends strength to the theory of the tubercular origin of pleuritis.

Coriveaud, on the other hand, reports a series of 27 cases followed for from eight years to twenty-five years, and found that none of them became tubercular after attacks of sero-fibrinous pleuritis. Such a series certainly confirms the view, held by probably the vast majority of observers, of the primary and non-tubercular origin of many cases of pleurisy with effusion.

In a paper read before the American Climatological Association, Dr. Vincent Y. Bowditch gives results of an investigation of the subsequent histories of 90 cases of pleuritis occurring in his father's practice in thirty years, and the conclusions he arrives at are that a large percentage of the patients who were afflicted with pleuritis, often in apparently chronic forms, recovered their health, and have not had a recurrence of the trouble nor developed subsequent pulmonary or other tubercular trouble.

In the last twelve years, of 11 cases of pleuritis with effusion seen in private practice, of which I have records, and the subsequent histories to date, 7 were purulent and 4 sero-fibrinous.

Of the 7 purulent, 6 have apparently recovered, and only 1 has developed tuberculosis; 5 of the 7 followed pneumonia. Of the 4 sero-fibrinous, 2 have apparently recovered and 2 developed tuberculosis. It must be admitted, however, that sufficient time has not yet elapsed to give these cases much value. The advocates of the tuber-

cular origin of pleuritis contend that many years may elapse before the pulmonary tuberculosis manifests itself—twenty-four years is the longest period I have found reported. It is very difficult to believe that there is any connection between the pleuritis and the subsequent pulmonary tuberculosis after so long a period, but it is not difficult to find observers who can believe much more improbable associations than this. Baumgarten believes that a child may be born with tubercle bacilli in some of its tissues, and that they may remain inactive for years, or even throughout life. Verneuil goes still further. He believes that the tubercle bacillus can exist throughout life in a parasitic form, and then manifest itself in a succeeding generation.

It will be noticed that six of my seven cases of empyema have apparently recovered. I have made no reference to a large number of hospital and dispensary cases observed, as it is impossible to get satisfactory subsequent histories, and the cases would have no value in connection with the subject of this paper.

The favorable progress of purulent pleural exudate is in accordance with the experience of von Ziemssen. He reports 18 cases of empyema; 15 of these were treated, of which 13 recovered. It should be stated that all of these cases of von Ziemssen's followed pneumonia. Many other observers have had a similar experience.

In 16 cases of dry pleuritis, more or less extensive, of which I have kept records, 5 have thus far developed pulmonary tuberculosis. These were cases occurring in private practice; hospital and dispensary cases are not included for the same reason that such cases were not included in my series of pleurisies with effusions, namely, unsatisfactory subsequent histories. Nor were cases included in which at the time there were unmistakable evidences of pulmonary disease. The observations extend over a period of fourteen years, and it may be objected that this is not a sufficiently long time to determine the question of a possible development of pulmonary tuberculosis later. Still, up to date, eleven are in such a condition as to justify the opinion that they have recovered.

During the past two years I have had observations made in 140 autopsies in which there were evidences of old or recent changes in the pleura, all cases being included in which such changes were found. These autopsies were performed on subjects from Bellevue and Charity Hospitals, the hospitals of the workhouse and almshouse of the city of

New York, and a few were cases of sudden death (coroner's cases) occurring outside of hospitals. There were changes in the pleura, more or less extensive, in nearly 80 per cent. of another series of autopsies, numbering 120, in which this was looked for, not including those cases where only a few fibrous threads connected the surfaces of the pleura. These changes consisted for the most part either in old adhesions, tubercular pleuritis, or recent acute pleuritis secondary to some other disease. These 120 cases were taken at random from autopsies made by Dr. Hermann M. Biggs at different hospitals in New York.

In the 140 cases 47, or 33.5 per cent., died from pulmonary tuberculosis, or else showed quite extensive pulmonary disease; of these, 9 presented unmistakable evidences of tubercular pleuritis. In 50, or a little over 35 per cent., of the remaining cases, cheesy or calcareous nodules were found in the lungs, making in all 97 cases, or 68.5 per cent., in which the lungs showed evidences of recent or old tuberculosis. Taking these two series as a whole, it is found that a little over 60 per cent. of the autopsies showed evidences of old or recent pulmonary tuberculosis, and of these less than one-half died of tuberculosis.

In 27 of the series of 140 cases the pleuritis was recent (in 10 of them there were also rather extensive old adhesions), and in 5 instances it was double and associated with pericarditis. In 5 there was anthracosis with fibrous new formations in the lungs, without tuberculosis, but associated with adhesions, as a rule, rather extensive. In 16 other cases there were changes in the pleura, in which the tubercular lesions were found. Of these, 5 were cases of tertiary syphilis, 2 were associated with new growths in the lungs, 5 others showed quite marked renal disease. In one rheumatic case there was acute pleuritis, pericarditis, and endocarditis; in one, chronic bronchitis and bronchiectasis; in one, an aneurism of the aortic arch, and one was a case of scleroderma with advanced heart disease.

In speaking of an old process, reference is made to the cheesy or calcareous nodules so frequently found in the lungs, and surrounded by fibrous tissue capsules. Recent investigations have clearly shown that in the vast majority of cases these nodules have resulted from a local tuberculosis; and that the cheesy masses generally, and the calcareous masses frequently, still contain the tubercular virus, and will, when inoculated, produce tuberculosis in animals. It should, also,

be distinctly understood that the nodules found in some of the cases were very small, not more than 3 mm. in diameter, and that in some instances not more than one nodule was found in the lungs. These have been included on the ground that they are exactly the same in nature as the larger masses, and if they are tubercular the question of size does not come into consideration. Fibrous cicatrices without cheesy nodules were not taken into consideration, although many of these unquestionably result from tuberculosis.

As regards the extent and distribution of adhesions, it was found that there was no constant relation between the degree of lung involvement and the extent of the adhesions. In some cases of very advanced disease the pleura showed only a few adhesions. This was especially the case where the process did not involve to any great extent the pleural surface. On the other hand, small nodules in one lung were not infrequently associated with extensive adhesions on both sides, and often the adhesions were more marked in the lung that contained no cheesy nodules.

The observations in these cases do not confirm the assumption that the adhesions are primary, and that they predispose to the development of a tubercular process in the lungs. The cheesy and calcareous nodules were generally found in the upper lobes, and near the apices, and were not infrequently found in other parts of the lungs also, especially in the upper portion of the lower lobes. When these nodules were found in the lungs, the bronchial glands usually showed similar changes macroscopically.

In the series of 140 cases only 9 showed unmistakable evidences of tubercular pleuritis, although 97 showed evidences of recent or old tuberculosis in the lungs, and 47 of these showed very extensive involvement.

Post-mortem researches seem to show that tubercular pleuritis is a rare disease, even though the lungs in many cases may be extensively diseased. Undoubtedly many cases of tuberculosis have their first manifestation in a pleuritis, and many cases of pleuritis are secondary to pulmonary tuberculosis. *Possibly* a pleuritis may favor the development of pulmonary tuberculosis by depressing vitality, by lessening the proper lung expansion, or by interfering with the nutrition of the adjacent lung; but more probably the same underlying cause produces both.

In the absence of evidences of tuberculosis in other situations there is no sufficiently precise clinical history to warrant an absolute diagnosis of tubercular pleuritis. If it is an effusion, the appearance of the fluid is not characteristic. Unless tubercle bacilli are found one cannot positively affirm its nature, and, as has been shown, the bacilli are very rarely found.

From my own researches, and a study of the researches of others, I offer the following conclusions:

First. If it be true that the very large majority of cases of pleuritis not traceable to ordinarily recognized causes are tubercular, we have no clinical method by which the fact can be proven, and the assumption that they are tubercular can only be by inference.

Second. If such a large majority of pleurisies, as many seem to believe, are of tubercular origin, recovery from tuberculosis of the pleura is much more frequent than from pulmonary tuberculosis.

Third. The prognosis in purulent pleural exudate is more favorable as to the subsequent development of tuberculosis, than in sero-fibrinous exudate.

Fourth. That even though, at an autopsy, evidences of pleuritis recent or old may be found, it does not necessarily follow that the pleuritis is tubercular because there are tubercular lesions in the lung.

Fifth. It is fair to assume from the frequent association, post-mortem, of recent and old changes in the pleura with pulmonary tuberculosis, that many of these changes are tubercular in origin, although at the time of death tubercles are not found in the pleura.

Sixth. In cases of pleuritis, although evidences of tuberculosis exist in the lungs, as shown by physical signs and by the presence of tubercle bacilli in the sputa, it does not necessarily follow that such cases of pleuritis are tubercular in origin, although they probably are.

Seventh. In cases in which pulmonary tuberculosis develops subsequent to a pleuritis, there is probably the same underlying cause for each.[1]

[1] I desire to acknowledge my indebtedness for much valuable assistance in the preparation of this paper to Dr. Hermann M. Biggs, one of the Curators of Bellevue Hospital and a director of the Carnegie Laboratory.

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